Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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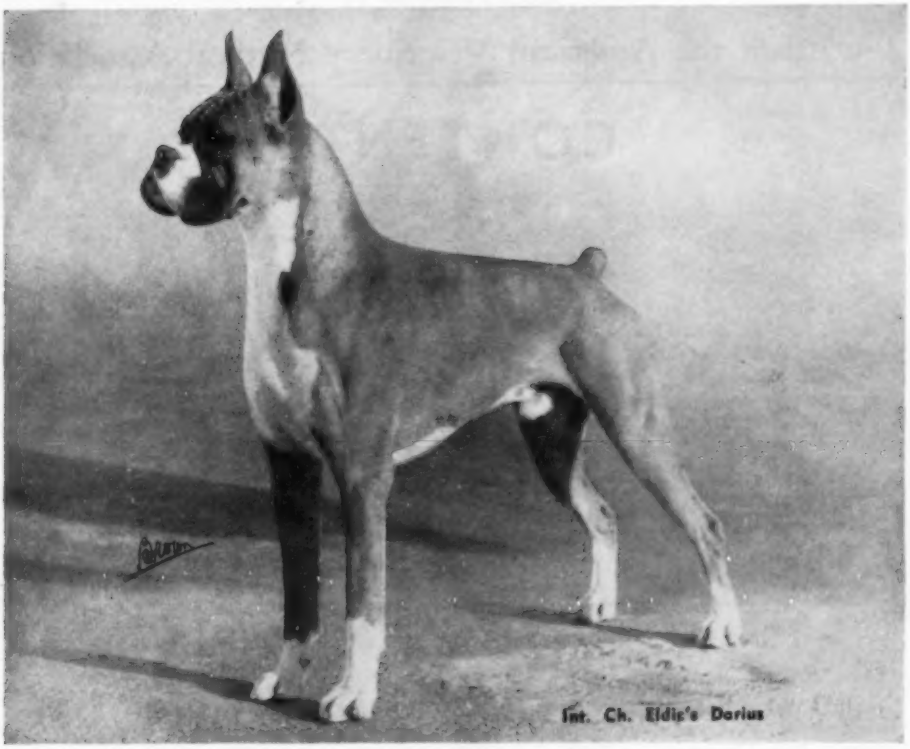

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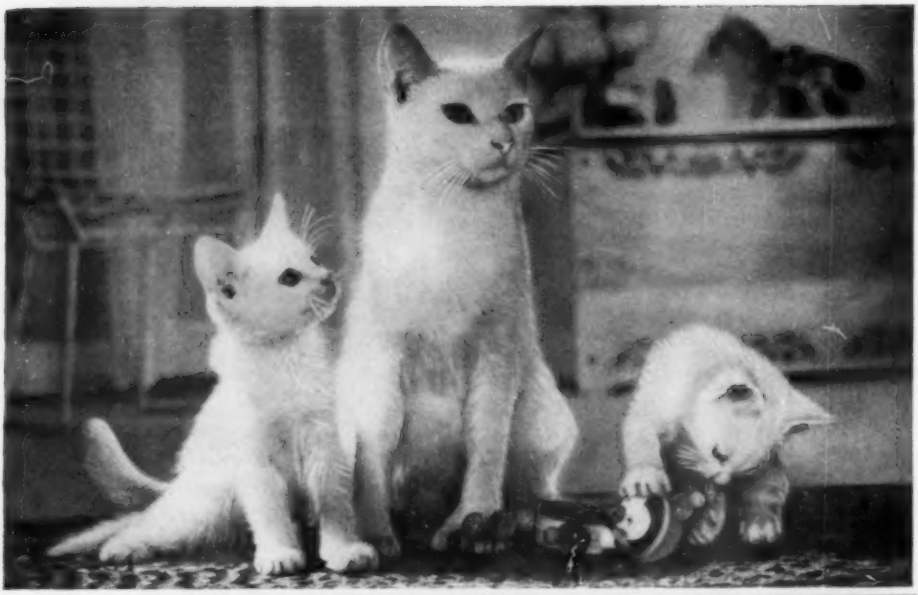

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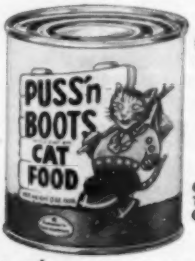

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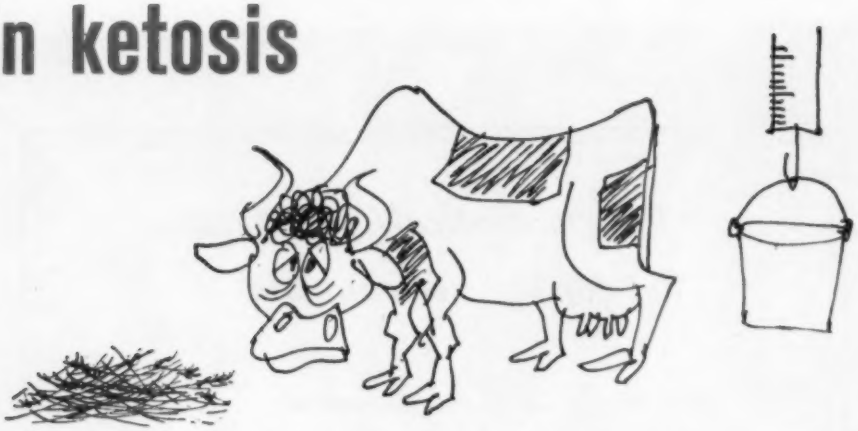

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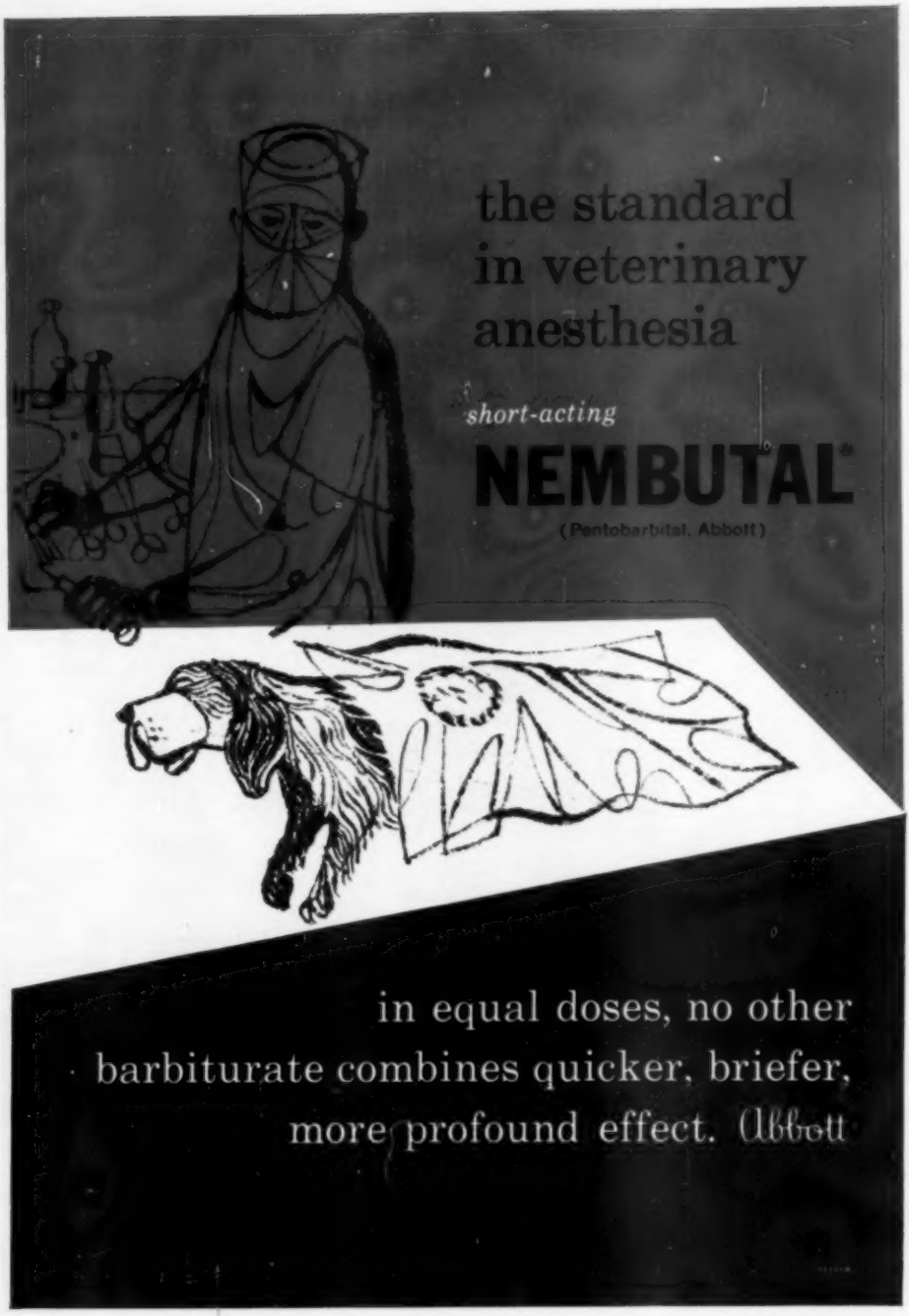
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AVMA Report

Internal and External Public Relations

Brig. General Wayne O. Kester,
President of the AVMA

Presented at the AVMA Public Relations Conference, Cleveland, August 18, 1957

I am one of many members who knows all too little about public relations. But here is an estimate of our situation I should like you to consider.

Just as urbanites see their veterinarian only as a doctor to pets or farmers as only livestock disease specialists, many veterinarians think of public relations only as publicity—the visible and most tangible part of public relations.

Public relations is far more than publicity. Basically, public relations is the communication of ideas. The hard part is in determining the ideas you wish to communicate and then employing the proper means to put the ideas across. The first part of any public relations program is deciding on an objective, then forming this objective into a concrete image for transmission. The basic idea in our case is service, which must be described in terms easily understood by those we serve. We serve the general public and all its parts: physicians, farmers, pet owners, and consumers—everyone. In the case of our Association, the primary public to be served is the member.

The general public is a composite of individuals with many different interests. Our profession is similarly composed. In both cases, there are many variations in type and degree of interests as there are individuals, yet there are certain common denominators.

Both the lay public and the Association member can best be served when there is an open, two-way communication of ideas. The common interests of our profession and the public are never static. In order to serve these interests, our profession and our Association must be aware of them and be kept informed of changes in them.

Fifty years ago with a more rural population, farm problems were understood by everyone and of concern to all. The concern remains but the understanding is lost because populations are shifting to urban centers. New generations are rising which think of milk only in bottles and that meat comes from a factory frozen or attractively packaged. They have little understanding of the effect animal diseases have on these products either in wholesomeness, quality, or price.

Our profession is changing also. In the past 50 years, practice has expanded from primarily the horse to all species. In the last 20 years, the profession has more than doubled in numbers and so has our Association membership. The uses to which our professional knowledge can be put has expanded. Entirely new facets of veterinary science are unfolding and old ones are expanding. Laboratories are seeking veterinary service to furnish better control of animals used in research. Zoos are finding that investment in veterinary service is sound economy. A knowledge of chemical effects on animal tissue used as food is a relatively new area requiring veterinary knowledge.

Before the public relations of our profession can be directed at the general public with maximum effect, the relations of our associations with the membership must be strengthened. Good external public relations requires a solid base of good internal relations.

Internal Relations

Constant surveys by our associations are necessary in order to find shifts and trends in the common interests of the public and the membership. These trends and shifts must then be reported to the members so that reaction can be determined and a course of action planned to further encourage or check the shift.

By maintaining a directory of our profession, by evaluating and disseminating information furnished by members, and by publishing a semimonthly Journal of scientific progress, news, and organization, the AVMA attempts to keep its members abreast of changes within the profession and allied groups.

The mechanics of a successful internal public relations program upon which the external program is based requires open and frequent communication from mem-

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bers of the profession to their local associations, from the local to the state or provincial, then to the regional and national association, and back again. Any break in the chain weakens the profession's public relations program internally and externally.

At every level, these reports of progress and changes can be turned to use for external public relations. This involves the communication of veterinary knowledge in a form usable by the general public. That is, in a form understood by the public and of a nature that is useful and helpful.

The objectives of an internal public relations program should then be for all associations to keep in touch with the problems of practitioners and apply this information through professional policies for the best interests of the client, the member, and the profession.

To obtain the information on which policy decisions are based, every member must be willing to contribute his experience, his views, and statistics to his closest organization. This group can then compile and forward such information to its parent group. The AVMA can then communicate the concensus of these reports to its constituents and so on until it reaches the membership.

Every member then has the opportunity to be aware of the advances and difficulties of the profession as a whole, and of steps proposed and action taken to continue advancement and to overcome difficulties.

Once such intercommunication is open and free-flowing, the profession can act as a team as close as two veterinarians in practice together. The profession can then move effectively toward strong, external relations with the general public.

The foregoing outlines the principles of a public relations system and the difference between internal public relations and external public relations.

You will see that we have a mechanism for an internal public relations system. It is a good mechanism and it is being improved. But no matter how perfect we build that mechanism, it is of no value if our individual members and constituent associations do not participate in it.

External Public Relations

The same is true of the external public relations system. It is a good system and is improving, but more improvement is needed. It, again, is built on the individual member and on service to the public by the individual veterinarian.

Now, how are we at the grass roots, the foundation building blocks in this system, so to speak? How are we carrying out our part? If we take a close look at ourselves, I wonder what role we might find. I wonder if we would find a fellow occasionally who gives nothing to his community except professional service. A fellow who feels that the world owes him a living and he is a little mystified when a good practice does not come to his door. He is not inclined to analyze why this practice evades him, and he does not seem to understand the workings of a two-way street. We certainly would not find many of these chaps because competition soon eliminates them—competition with other veterinarians or agencies who are more progressive and forward-looking, especially with respect to public relations.

Then I wonder if we might find another type—the fellow who thinks that, because he works among farmers, he must look like a tired dirt farmer at the end of a hard day's work in a cornfield. The chap who delights in wearing rumpled old clothes. The rugged fellow who just would not be seen in public wearing a business suit like other business and professional men in his town. This is ironic because our clients dress up a bit when they go calling, even when they make a business call on us.

I often think of the first veterinarian I ever knew as a youngster in my home town. He was always immaculately dressed even when he was hard at work. In the eyes of the public, he was the equal of the banker, the lawyer, or any other citizen, including his honor, the mayor; in fact, he was the mayor. He thought it paid to be neat, clean, and circumspect about dress, appearance, and conduct before his public and, from my observation, it still pays. Also from my observation, we have very few of these careless, sloppy characters and even though they generally provide excellent professional service, competition rapidly squeezes them out. Perhaps this is well because they do us very little good from a public relations standpoint.

Then there is another type that I wonder

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SUMMIT, N. J.

1. Karmin, L. R.: N. Am. Veterinarian *36*:846 (Oct.) 1955. 2. Earl, A. E.: J. Am. Veterinary Med. Assoc. *129*:227 (Sept. 1) 1956.

if we might not find occasionally. This one worries me. He is the chap who fails to go the limit in providing service for his clients. The fellow who is unaware that he is obliged to give his client a complete service and does not follow through. He does not seem to realize that in this world of modern medicine his clients do not expect him to have all the answers but they do expect him to obtain consultation and get the answers he does not have. He is, in fact, morally and professionally obligated to do so. Here is a really dangerous character because, when he throws up his hands and tells his client there is nothing more that can be done on a case, he, in fact, forces the client to go elsewhere, as most clients leave no stone unturned in solving their problems. Consequently, he may wind up at a state or federal service seeking advice and receiving a sympathetic ear from some source outside our profession. We don't have many of these characters in our midst. However, probably more serious public relations problems or incidents stem from this source than any other.

Then we have the fourth type. This I know. The fellow who enjoys providing service—any kind of service. The fellow who is not only a staunch and competent doctor of veterinary medicine and well known as such, but he is also chairman of the school board, commander of the local American Legion, a leader in the chamber of commerce, and a real part of his community.

He knows the world does not owe him a living. He sells his profession and himself to the public, seemingly without being aware that he is so doing. This is the kind of chap we must have as a foundation to build a public relations program and this is the kind of a fellow we have in vast numbers.

So we have the foundation building block upon which the internal public relations machinery is built. These in turn serve as a base for the external PR mechanism. The three combined form our public relations system. It is an excellent system that will work as well and do as much as we, as individuals, make it.

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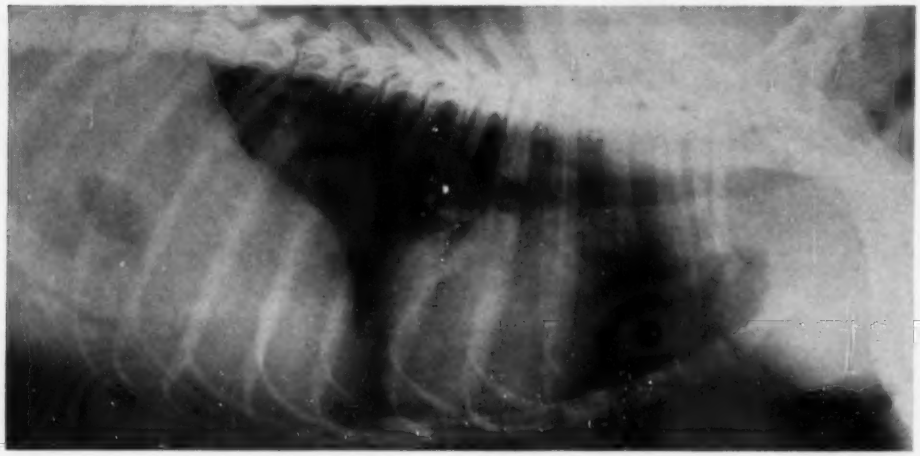

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Control of Leukosis in Fowl

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Ithaca, New York

EXPERIMENTS TO DETERMINE the feasibility of breeding strains of fowl that are resistant to leukosis have been continuous in this laboratory since 1935. The work was undertaken because it was evident that the usual orthodox measures for controlling disease were completely ineffective against leukosis while, at the same time, familial differences in resistance and susceptibility to the disease were evident in the records of any pedigreed, exposed flock.

The term "leukosis," as used in this paper, includes the neural, visceral, and ocular forms of lymphomatosis which have been grouped together as different expressions of the avian leukosis complex. Other neoplasms of this complex were rarely encountered. We are aware that other classifications are now being proposed. If there are two separate diseases, neural lymphomatosis and visceral lymphomatosis, this paper deals with both. Both cause serious loss to the poultryman. Since the measures effective against either one are the only ones known to be effective against the other, no apology is needed, in this discussion, for continuing to treat them together under the label that they have carried for over 20 years.

While the primary objective of this experiment was to determine the feasibility of developing strains resistant to disease, it was recognized from the beginning that this would be futile if the resistant fowl could not lay well or be just as profitable as those not specially selected for resistance to disease. Accordingly, selection has been practiced not only for resistance to leukosis, but also for resistance to other diseases and for such economic traits as high egg production, adequate egg size, body size, and ability to reproduce.

From the Department of Poultry Husbandry, New York State College of Agriculture, Cornell University, Ithaca.
Presented before the General Session, Ninety-Fourth Annual Meeting, American Veterinary Medical Association, Cleveland, Ohio, Aug. 19-22, 1957.

PROCEDURE

Our procedure has been to ensure natural exposure of each generation during the critical first few weeks after hatching, and then to determine subsequent mortality from the disease up to 500 days of age. All breeding birds have been evaluated by the viability of their offspring, of their siblings, or of both. To that end, no culling of unthrifty birds has been done in the families under test.

When selecting breeding stock on the basis of egg production, we searched the records not only for the best sire families, and the best dams' families within them, but also for the best individuals within those families. Viability and performance were measured in females only; about 2,600 have been tested annually. They represent three distinct lines, two of which are bred for resistance to leukosis, and the third for susceptibility to that complex.

The birds under test were given neither intraperitoneal nor oral inoculations of any kind, but were left to contract the disease by whatever channels cause natural infection. When the experiment was begun, a high level of leukosis in the Cornell flocks seemed to ensure adequate exposure for differentiation of resistant and susceptible families. A few years later, it was discovered that close proximity to adult stock during the first two weeks increased considerably the severity of exposure, and thereafter it was made routine practice to brood all chicks under test as near as possible to adult laying birds. Attendants walked regularly from old hens to chicks, and disinfection of quarters for all birds was completely abolished.

Not only was this natural exposure adequate for the selection that was practiced but, as experiments have shown,[1] it provided a better measure of genetic resistance to the disease than could have been obtained if the birds had been inoculated with

some standard dose of supposedly infective material.

Progress in the initial years of the experiment was slow, partly due to the inevitable complications and compromises necessary when breeding for several objectives at once. Had we been concerned only with developing a strain resistant to leukosis, and not with its ability to lay well and to lay large eggs, the problem would have been much simpler. Furthermore, it was difficult to get enough sires that would transmit good resistance along with other desirable qualities. Eventually, a system of "multiple shifts" was adopted, by which we were able to test three times as many cockerels per year in the same facilities.[9] After that scheme was put in operation, progress was comparatively rapid.

RESULTS

Progress at various stages in this experiment has been reported in detail elsewhere.[4,5,7] The records for the last three generations that have completed their tests (table 1) show that, in the two resistant lines, mortality from leukosis is now at a negligibly low level. The average for the K resistant line was less than 1.0 per cent and for the C resistant line about 1.5 per cent. At the same time, mortality from the three forms of leukosis combined in the susceptible line was 38.8 per cent. Much of the leukosis in the susceptible line was of the neural form. In spite of high mortality from that type, which removed many birds before the age when the visceral form is more common, the incidence of visceral leukosis in that same strain was 5.6 times as high as in one of the resistant lines and 14 times as high as in the other. The comparatively high incidence of neural lymphomatosis in our susceptible stock has led some investigators to state that we are dealing only with that form of the disease. Clearly, that is not the case. Both neural and visceral lymphomatosis have been continuously present on our premises since the experiment began. Evidence from different sources (which can not be given in detail here) has shown that the flock has been exposed annually to whatever agents cause the visceral form as well as to those causing the neural type.

The results can be summed up by stating that for several years the incidence of leukosis in our resistant stock has been so low that losses are negligible, and further selection against it is difficult because of the very low incidence of the disease. Were it not for the continuing high incidence of leukosis in our susceptible strain, one might suppose that the disease had disappeared.

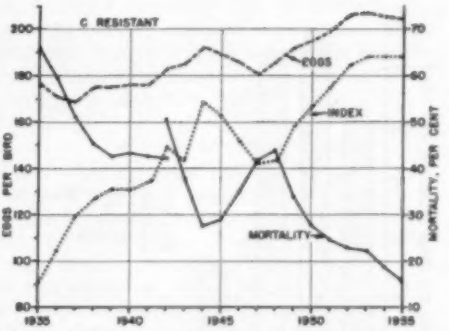
The effect of selection for viability over the years is shown (graph 1) in the performance of the C resistant strain from 1935 up to the present time. From a high figure of about 66.0 percent in 1935, mortality from all causes has gradually dropped to 15.5 per cent in the generation hatched in 1955—the latest to complete its test. These figures apply to unculled populations for the period from 6 weeks to 500 days of age. At the same time, the average egg production of surviving birds has risen from 177 to above 200 for the last five generations. These records apply to a laying period of about 335 days only and are not to be compared directly with figures for egg production of birds tested for a full laying year of 365 days.

TABLE 1—Mortality (%) from Leukosis and Other Neoplasms in Three Strains of White Leghorns from 43 to 500 Days of Age

Strain	Year	Females (No.)	Leukosis†			Other neoplasms	Total neoplasms
			Neural	Visceral	Ocular		
K resistant	1953	1,405	0.3	0.3	none	0.1	1.1
	1954	1,027	0.5	0.2	none	0.1	0.8
	1955	1,084	0.4	0.4	none	0.3	1.0
	Average*	1,172	0.4	0.4	none	0.2	1.0
C resistant	1953	803	0.4	1.2	0.1	0.9	2.6
	1954	1,171	0.3	1.6	0.1	0.3	2.3
	1955	793	0.5	0.3	none	1.1	1.9
	Average*	922	0.4	1.0	0.1	0.8	2.3
Susceptible	1953	663	32.0	7.1	0.2	1.7	40.9
	1954	742	45.0	5.9	1.1	1.5	53.5
	1955	747	21.4	3.9	none	2.8	28.1
	Average*	717	32.8	5.6	0.4	2.0	40.8

*Unweighted. †Includes under visceral, a few cases of erythroleukosis, myeloid leukosis, and myelocytomatosis.

Some idea of the value of such a disease-resistant strain is given by the production index (graph 1). It is the same thing as the average egg production for all hens housed. As it can be raised either by higher viability or by better egg production, the index has the advantage of combining in a single measure the contributions of both factors to the economic value of the flock. In the C resistant strain, the index has risen from 90 in 1935 to 187 for the flock of 1955.



Graph 1—Reduction of mortality, increase in egg production, and rise of the production index in the C resistant strain of White Leghorns. The temporary rise in mortality in 1947-1948 was caused in part by diseases other than leukosis. All lines are smoothed by use of three-year moving averages except for terminal points.

TESTS OF RESISTANT STOCK

As facilities for adequate tests of our resistant stock against the highly improved strains of other breeders were not available, comparisons were made by entering random samples of the resistant strains in the New York State Random Sample Test. This was done for 1953, 1954, and 1955. In that test, a sample of unpedigreed eggs from flock matings was given each year to the supervisor, and thereafter control of the birds and records of their performance were completely out of our hands. Those that died were diagnosed along with all other birds from the Random Sample Test at the New York State Veterinary College.

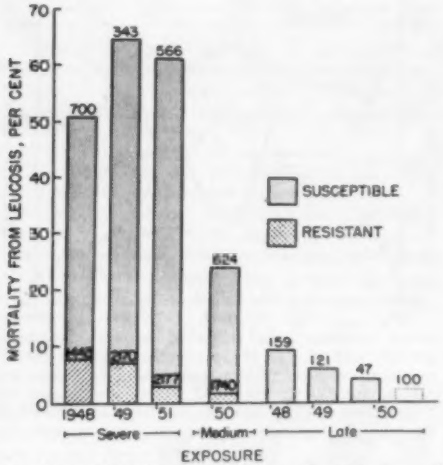
The record for three years showed that, among the 25 good strains which had been entered for all three years, the Cornell resistant stock ranked lowest in mortality from all causes, lowest in mortality from neoplasms of any kind (including leukosis of all kinds), and second to the highest in income per chick started. Some of the other strains laid at a better rate, but returned lower profit per chick started because higher mortality reduced the total number of eggs laid by the flock.

These results showed that it is quite possible by selective breeding to develop stock so resistant to leukosis that, even under conditions of fairly severe exposure, mortality from that disease can be reduced to an almost negligible level. We are now reporting on 21 years of selection, but it does not take that long to develop highly resistant stock. With more extensive facilities for testing sires, and with present knowledge about exposing the birds under test, anyone should be able to develop a high degree of resistance in five generations.

CONTROL BY ISOLATION

After seven years of this experiment, it was discovered that half of the pullets raised each year had been getting a milder exposure to leukosis than the other half.[9] Odd-numbered hatches had been kept for their first two weeks in one brooder house and even-numbered hatches in another. Thereafter, both went to the same raising range. The odd-numbered hatches were experiencing up to four times as much leukosis as the even-numbered ones and the only factor that could be found responsible was the closer proximity of the former to adult birds. Further tests indicated as had earlier isolation experiments,[10,11] that by brooding the chicks away from adult birds subsequent mortality from leukosis could be greatly reduced. Complete isolation is impracticable for most poultrymen, but our findings indicated that a partial isolation, a separation of the chicks from adults by even 120 ft., could make a difference.

In further studies, it was found that even highly susceptible stock could escape severe losses if the chicks were brooded in complete isolation and not returned to infected premises until they were 5 months of age. From such experiments[6] (graph 2), it became clear that mortality from leukosis could be greatly reduced, either by developing genetically resistant stock, or by preventing susceptible stock from becoming infected during the critical first few weeks after hatching. The latter procedure is more practicable for the majority of poultrymen. However, even partial isolation is sometimes difficult to provide and the need for genetically resistant stock still remains. Many good breeders are now developing



Graph 2—Mortality from leukosis (to 500 days of age) in four generations of White Leghorns. Figures above the columns give the numbers of females in the various groups. The protective value of genetic resistance is shown by comparisons between the resistant and susceptible strains when exposure was severe, or medium. The protective value to a susceptible stock of isolation at early stages is shown by comparisons in 1948 and 1949 between such birds exposed severely (right after hatching) and their full sisters not exposed until 160 days later. The short, dotted column at the right represents genetically resistant birds exposed late, among which not 1 died of leukosis.

highly resistant strains with which to meet that need. If hatcherymen would use such stock to head their flocks, and if poultrymen would provide all the isolation they can manage, mortality from leukosis would be greatly reduced.

RESISTANCE MAINTAINED IN OTHER LOCALITIES

To determine whether or not resistant strains developed in one locality would maintain their genetic resistance to leukosis when transported to other areas, our two resistant strains of White Leghorns and one similarly developed at the Alabama Experiment Station were exchanged and maintained under controlled conditions at the two experiment stations. The results showed that all strains tested maintained the same comparatively high resistance to leukosis that they had demonstrated in their home states,[12] some 900 miles apart. The same thing has been demonstrated by strains bred for resistance in other states which have been tested in the New York Random Sample Tests.

TRANSMISSION THROUGH THE EGG

The possible influence of transmission of the causative viruses through the egg was tested experimentally in two years by sending to an isolated farm certain complete hatches of our genetically susceptible strain. These were kept in isolation until about 5 months of age and then returned to infected premises. There was no evidence that transmission through the egg of pathogens causing leukosis is of importance in practice.[2] Among 280 pullets of this highly susceptible stock that were reared in isolation, only a single case of leukosis developed before the birds were brought back to the infected premises. After that exposure, subsequent mortality from leukosis was 9.4 per cent in one year, and 5.8 per cent in the next but, among their full sisters which had been exposed right from hatching, the corresponding figures were 54.0 and 64.0 per cent. If leukosis were transmitted through the egg, the isolated birds should have been infected like the others.

This does not mean that the causative virus can not be put in the egg or transmitted through it, but it does indicate that in practice such transmission is not important. It has been stated that our failure to find it so is attributable to the fact that we are (supposedly) working principally with neural lymphomatosis. However, in other stock raised on our premises, the incidence of visceral leukosis is much higher than in our genetically resistant strains of White Leghorns. Other evidence that any transmission through the egg is unimportant has been summarized.[1]

SUMMARY

By selection of genetically resistant stock, two strains of White Leghorns have been developed in which mortality from leukosis, in birds adequately exposed, is now 0.8 to 1.5 per cent. At the same time, in a strain bred for susceptibility to the complex, average mortality from leukosis was 38.8 per cent, much of it of the neural type.

It has been shown that both genetic resistance and ability to lay can be raised concurrently, with the resultant strain having higher viability and greater productivity.

A considerable measure of control can be effected by brooding the chicks in isolation,

as far as possible from older birds, during the critical first few weeks after hatching.

Experiments with isolated stock indicate that any transmission there may be of causative agents through the egg is relatively unimportant in comparison with the degree of genetic susceptibility and the severity of exposure to the disease after hatching.

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Gas Gangrene in Man.—Of 42 patients with clostridial myositis (gas gangrene), *Clostridium welchii* was present in all but three, alone or in combination with other clostridia. The interval between injury and lesions was as short as six hours in wounds associated with gross devitalization and contamination of muscle. The average incubation period was 53 hours. Pain was the first and most important symptom. The temperature in the early stage varied from 97 to 107 F. Radical treatment such as multiple incisions for decompression, excision of the involved muscles, or open amputation should be done as soon as possible, along with penicillin and tetracycline therapy.—*J.Am.M.A. (Sept. 21, 1957): 291.*

Hormone Studies with Siamese Twin Rats.—To explore the effect of hormones on diseases such as cancer, at the University of Wisconsin, 30-day-old rats (too young to produce sex hormones) are joined at the shoulder. Later, when the ovaries of 1 rat are removed, the "brake" on its pituitary is released and it produces an abnormal amount of gonad-stimulating hormones, causing the ovaries of the uncastrated "twin" to grow rapidly. Various hormones are tested by injecting them into the castrated twin. The natural female hormone (17-*beta*-estradiol) produced the best "brake" on the pituitary; testosterone was next best; and progesterone was much weaker. The testing continues.—*Lab World (June, 1957): 304.*

Dangers in Steroid Hormone Therapy.—Steroid therapy was considered directly responsible for the death of at least 11 human patients. It had suppressed or masked evidence of infection in at least six and had caused adrenal failure in others.—*J.Am.M.A. (July 27, 1957): 1502.*

Actinomycosis in Man.—Of 37 patients with actinomycosis, the primary lesion was in the cervicofacial tissues in nine, the lungs in five, and the abdominal tissues in 23. The present standard therapy is 10 to 20 million units of penicillin daily in a continuous intravenous drip for 30 to 45 days, then wide surgical excision. Of those given this treatment, 23 per cent died of the disease.—*J.Am.M.A. (Aug. 3, 1957): 1614.*

Treating Leptospirosis in Man.—There is no satisfactory treatment for infections caused by *Leptospira icterohaemorrhagiae* (Weil's disease) or *Leptospira canicola* (Stuttgart's disease) in man. Several antibiotics are moderately effective against leptospiras *in vitro* but, unless given during the first few days of illness, their effect *in vivo* is doubtful.—*J.Am.M.A. (Sept. 7, 1957): 117.*

Recent Studies on the Natural Transmission of Visceral Lymphomatosis in Chickens

B. R. BURMESTER, D.V.M., Ph.D.

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ONLY OCCASIONALLY does visceral lymphomatosis (VL) occur in flocks in such proportion and at such time intervals as to suggest that it is a contagious disease. Almost invariably, it causes death at a low but continuous rate during the adolescent and adult life of the chicken. The year-to-year fluctuation on any one farm usually is not great, although multi-year cycles of greater amplitude do occur. Earlier experiments indicated that VL was a nontransmissible neoplasm. However, one can not ignore the overwhelming accumulation of data which indicate that VL is caused by a biologically reproducible entity that is naturally transmitted by contact and from parent to offspring through the egg.

This paper will be confined almost entirely to recently published data and will be concerned only with visceral lymphomatosis. During this period, little that is new concerning the transmission of neural or ocular lymphomatosis has been reported.

EGG TRANSMISSION

The first evidence that the causative agent of VL is transmitted from the dam to the offspring was furnished by experiments reported in 1954.[13] When inoculums from livers of 39 embryos from eggs laid by normal appearing hens were tested for the presence of the VL virus, by the intraperitoneal injection of susceptible day-old chicks, approximately one third gave evidence of VL virus in infectious amounts. The agent was also demonstrated in the livers of newly hatched chicks.

In subsequent experiments,[7] a study was made of the shedding of the virus in eggs laid over a two-year period by 22 normal appearing White Leghorn hens from an infected flock, and also in the eggs of their progeny laid during a one and one half-year period. Of the 22 hens, 17 were of two susceptible inbred lines and 5 were from a line selected for resistance to lymphomatosis. All chickens were raised together in an effort to obtain a uniform exposure for the various lines.

Tests, when the hens were approximately 1 year old, revealed that 15-day embryos from 16 of the 17 hens of susceptible lines contained enough virus to produce a significant incidence of VL in inoculated chickens, whereas only 1 of the 5 hens from the resistant lines shed the virus into her eggs. Similar tests, conducted three to six months later, indicated some variation in the shedding of the virus but a generally high level of virus in eggs was evident for hens of the two susceptible lines.[9] However, when the same hens were 2 and 3 years old, the levels of transmitted virus were much lower.

Similar tests on eggs laid by progeny (8 to 9 mo. old) of the hens of the two susceptible lines, showed that 9 of 14 shed significant levels of virus in their eggs.[9]

Only 3 of the 22 hens died of lymphomatosis—1, a week after the eggs for the first test were laid, and the other 2 almost a year later. Eggs, infected naturally or by inoculation, hatch well and the chicks have high viability.[6,14]

These results provide conclusive evidence that the VL virus may be deposited in eggs and infect the embryo, while the hen, the embryo, and the chick appear normal. The VL virus propagates in embryonating eggs;[14] hence, the response obtained with 15-day embryonic liver was the result of virus deposited and then propagated during 15 days of incubation. In addition to the state of infection of the dam, it would appear that genetic constitution and age are important factors in determining the extent of virus deposition in eggs.[9] Within any given infected flock, young hens of susceptible lines are more apt to shed virus into their eggs than are older ones and those of resistant lines.

Although both neural and visceral lymphomatosis occurred in the flock that supplied the eggs for the infectivity tests, VL alone was observed in the chicks inoculated. These results give further evidence that neural and visceral lymphomatosis are not

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caused by the same agent and indicate that the neural form may not be egg-borne.

Since the amount of virus in livers of 15-day embryos of certain "carrier" hens was sufficient to cause a high incidence of tumors when distributed by intraperitoneal injection to 12 susceptible test chicks, one would suspect that the progeny from the same carrier hens would have an equally high incidence of VL. That this is not the case is shown by the results obtained with two sets of progenies. They were obtained from the 22 dams that had been classified, according to the relative incidence of VL in the test chicks, as 12 heavy shedders, 5 light shedders, and 5 nonshedders. The progeny of one group[7] were hatched and raised together in regular incubators and brooder units while those of the second group[6] were hatched and brooded in special family (progeny of single male and female mating) isolation units.

The occurrence of VL in the first group of progeny was generally higher than in those raised in isolation; however, in both groups there was no apparent relationship between the amount of infectious virus in embryos of certain dams and the occurrence of VL in the progeny of the same dams. Thus, many hens which had been classified as heavy shedders had progeny none of which developed VL; whereas, others classified as nonshedders had several progeny which developed lymphomatosis. It appears that the presence or absence of the VL virus in the embryo is not the most important factor in determining whether chickens with such exposure develop VL.

It is well known that inherent resistance plays an important role in determining the occurrence of lymphomatosis; however, it has not been generally recognized that antibodies transmitted from parent to offspring via the egg may also play an important part. The significance of this influence is suggested[4,5,11] in recent experiments. Hens that had received repeated injections of the VL virus developed a high titer of neutralizing antibodies, and the progeny obtained after the immunizing injections were much more resistant to challenge inoculation than were the progeny of the same hens before injection. Neutralizing antibodies were also found in serum of day-old chicks of the immunized hens. Similar levels of neutralizing antibodies were found in chicks from hens naturally infected and shown previously to have shed the VL virus into their eggs.[18]

Several live-virus vaccines widely used on chickens are propagated in embryonating chicken eggs. Since VL occurs as an inapparent infection in many chickens of most flocks, it is probable that certain embryos used in the manufacture of vaccines had been infected with VL. The possibility of transmitting lymphomatosis by this means was investigated in a series of four experiments in which both specially and commercially prepared Newcastle disease vaccines were used.[10] Chickens of a highly susceptible line, vaccinated with one of the two commercial vaccines tested, had a significantly greater incidence of VL than the nonvaccinated control group. Similarly, in two additional experiments, chickens that had received Newcastle disease vaccine, prepared from embryonating eggs laid by hens that had shed the VL virus, developed a higher incidence of VL than chickens that received vaccine prepared from eggs laid by noninfected hens.[10] Thus the data indicate that the use of VL-infected eggs in the preparation of live-virus vaccines may be a significant factor in the transmission of this disease.

CONTACT TRANSMISSION

The role of the egg-transmitted virus becomes apparent when we examine the results of contact transmission experiments. Numerous investigators have shown that chicks of a relatively disease-free source, when raised in the same brooder units with chicks of the same age from an infected flock, develop an incidence of lymphomatosis which is usually higher than in the progeny of the infected flock.[2,6,15,17] The VL virus was demonstrated to be present in the debris of an incubator that had been used to hatch chicks of an infected flock.[6] When chicks of susceptible disease-free parents were hatched in the same incubator with chicks from infected parents, an increase in lymphomatosis occurred in the chicks of the disease-free susceptible source.[6]

Transmission of the disease from inoculated chickens to noninoculated penmates has often been reported. Factors which directly influence the rate of contact transmission include: intimate contact of chickens of similar age, young age at exposure, and a high proportion of infected chicks.[2]

Experiments have indicated that chick-

ens may be infected through any of the natural body openings. Disease was produced, following inoculation with the strain RPL-12 VL virus, by different routes in the following descending order of effectiveness —tracheal, nasal, cloacal, conjunctival, oral, and aerogenic.[3]

Studies showed that oral washings from naturally and experimentally infected birds were highly infectious.[1,8] Oral washings of certain normal birds of an infected flock and their day-old progeny likewise were infectious, although somewhat less so than the washings from lymphomatous chickens.

Experiments on the shedding of VL virus in the saliva revealed that, when the chicks were inoculated at 1 day of age, highly infectious oral washings were obtained as early as 10 days and, with little variation, remained so for as long as 180 days.[8] Similar tests on chickens kept in direct contact with the inoculated chickens showed that the oral washings collected from birds 90 days of age were the first to contain significant levels of virus and, at 180 days, the oral washings were as infectious as those from the inoculated chickens. Of the 12 inoculated birds from which oral washings were taken, 7 died of VL when 98 to 236 days of age, whereas 4 of 12 contact birds died when 206 to 212 days of age. This suggests that chickens shed highly infectious levels of VL virus in their saliva soon after they have become exposed. When the exposure is definite and massive, large amounts of virus appear within ten days, although deaths may not occur until several months later. When the exposure is indefinite, as by contact exposure, a longer period is required for the average virus levels in the saliva to attain highly infectious concentrations.

Infectious levels of VL virus were found in feces from some normal appearing hens and lymphomatous chickens. Subsequent experiments showed that the infectivity of a virus preparation may be lost when it is mixed with the droppings of certain birds, thus indicating the presence of a neutralizing or inactivating principle in the feces.

DISCUSSION

The presence of infectious levels of the VL virus in the saliva of baby chicks, when infected from the egg or by contact during the first three months of the brooding period, is highly significant, especially in view of the fact that the chickens remain in apparent good health for long periods thereafter. Feces may also be a source of infection, although it probably plays a lesser role since the apparent concentration of virus appears to be lower and less regular in its presence.

The data indicate that the virus of VL is spread by both normal appearing and diseased hens via the egg, the saliva, and the feces. Baby chicks hatched from infected eggs contaminate the incubator by disseminating dried extra-embryonic fluids, feces, and other hatching egg debris. Chicks from infected eggs shed virus in their saliva and feces during the brooding period and contaminate the feed, water, and equipment of the brooder unit, thus providing an effective source of infection for susceptible penmates.

Experimental administration of virus by the oral and respiratory routes has resulted in VL; hence, it is probable that these routes are portals of entry for naturally disseminated virus. Results of one experiment have incriminated the common drinking fountain as a possible means of disseminating the VL virus in the brooder unit.[18] It was found that the extent of transmission between chicks inoculated intraperitoneally and noninoculated chicks of the same hatch, when brooded in a pen with a rapidly flowing water fountain, was only about one third as great as the transmission between birds similarly treated but raised in a pen with a static water fountain. Virus placed directly in a nonflowing water fountain of a third pen produced as much VL as occurred in the contact chicks of the nonflowing water pen. These results indicate that chicks infected at 1 day of age shed enough virus in their saliva to cause an infectious contamination of a reservoir type drinking fountain. Furthermore, it would appear that the contaminated water of such fountains was responsible for the spread of infection to susceptible, noninfected penmates. It is likely that under natural conditions this mechanism of the spread is operative, since it has been shown that day-old chicks of naturally infected hens shed virus in their saliva.[18]

SUMMARY

It has been demonstrated that a high proportion of hens of a flock may have an inapparent infection with the virus of visceral lymphomatosis (VL). Such hens may re-

main healthy for long periods and shed infectious levels of virus into their eggs, saliva, and feces. Hens with inapparent infection may have circulating antibodies which are also transferred to their eggs, giving some protection to their progeny. However, such progeny shed virus in their saliva and feces, contaminating the incubator and brooder units. This provides a source of infection for the more susceptible birds, in the same unit, the progeny of hens without appreciable antibody levels.

When the virus is applied to the mucous membranes of any of the natural body openings, VL will result. Experiments indicate that drinking water may play an important role in transmission within the brooder unit.

The feces and saliva of infected adult chickens seem to be unimportant sources of infection in the natural transmission of this disease.

Live-virus vaccines containing chicken embryo materials may be contaminated with sufficient VL to cause an increase in the occurrence of the disease in the chickens vaccinated.

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Effect of Vaccines for Visceral Lymphomatosis.—When chickens less than 1 year old were given several injections of fully virulent virus of visceral lymphomatosis, or two injections of an adjuvant preparation, their progeny were much more resistant to inoculation with the same virus than progeny of untreated hens. Killed preparations of the same virus provoked an immune response in the hens and a resistance in their progeny which was significant but less than that obtained with live virus preparations. The progeny also showed a marked reduction in the development of osteopetrosis.—*Vet. Bull. (Aug., 1957): Item 2451.*

Effect of Temperature on Newcastle Disease Vaccines.—When Newcastle disease occurred in chickens just after they were vaccinated with a formalized adsorbed chicken embryo vaccine, samples of the vaccine stored at 37 C. and at room temperature (28 to 30 C.) were tested. Chickens vaccinated with a vaccine kept at 37 C. remained healthy while those given vaccine kept at room temperature for varying periods developed the disease and died. —*Vet. Bull. (Aug., 1957): Item 2405.*

Incubation Period of Rabies in Man.—Of 263 cases of rabies in man reported in

Taiwan during a four-year period, the incubation period was known in 235. Of these, it was one month or less in 33.6 per cent; one to three months in 44.3 per cent; three to six months in 11.5 per cent; and more than six months in 10.6 per cent. The disease showed no striking seasonal variation and no wild animal transmission was reported; the source of infection was dogs (95.9%); cats (3.3%); and swine (0.8%).—*J. Formosan Med. A. (Nov., 1956): 548.*

Shackling as a Factor in Ham Hip-Joint Hemorrhage—An Abstract

Studies conducted in four meat-packing companies indicate that approximately 80 per cent of 4,416 hams examined had coxofemoral hemarthrosis (internal hemarthrosis—fig. 1). In approximately 10 per cent, the joint capsule had been ruptured and the bloody synovial fluid had escaped into the intermuscular septums (external hemarthrosis—fig. 2 and 3). This condition causes an economic loss to the meat industry, as the defect results in an unpalatable product unless extensive labor is used to trim away the bloody meat. In many instances, the defect is not visible on the surface of the ham. In these, the consumer finds the defect when carving the ham and either returns it as defective, or buys another brand of ham, or eats beef or poultry for awhile in place of ham.

From the results of several experiments, four lines of evidence clearly indicate that shackling is primarily responsible for the external hemarthrosis. First, 95 per cent of them were from the shackled leg. Second, fewer were found in a plant where shackling was eliminated (1.9% as compared with 10%). Third, shackling by the forelegs resulted in fewer external hemarthrosis (1.4%). Fourth, they were absent when the hogs were not suspended by the hindlegs (fig. 1).

Two lines of evidence indicate that the gambreling procedure, whereby the carcass is suspended by a gambrel inserted in the hindlegs, is largely responsible for producing the internal hemarthrosis: (1) the complete absence of internal hemarthrosis when the hogs are neither shackled nor

Fig. 1—Opened hip joints from hogs shackled and gambreled (left) compared with hip joints from hogs neither shackled nor gambreled (right). Note presence of blood (internal hemarthrosis) in the synovial fluid of the hip joints from the shackled and gambreled hogs.

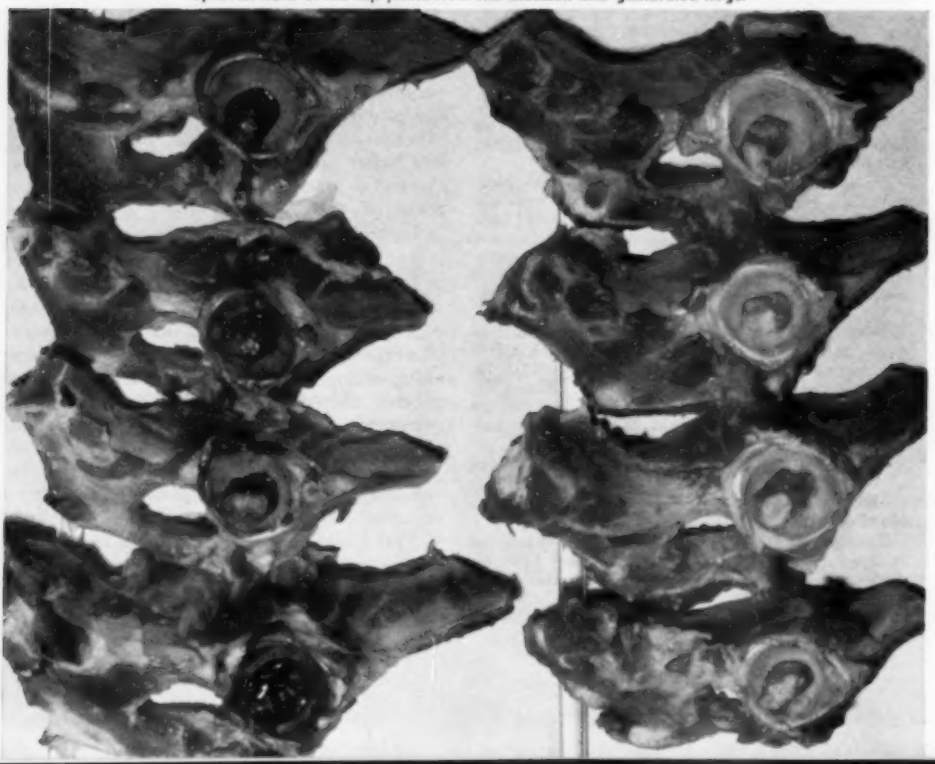

Fig. 2—Photograph of the medial surface of a ham showing the area where severe type of external hip joint hemorrhage appears on the surface of the ham (arrow).

gambreled (fig. 1); and (2) the elimination of shackling did not result in an appreciable reduction in the incidence of internal hemarthrosis.

As a result of these studies, a horizontal sticking and bleeding conveyor was developed. This conveyor eliminated the shackling process. In an extensive series involving 2,300 hams, it was found that the incidence of external hemarthrosis was substantially reduced (1.9%) as compared with a plant which shackled the hogs prior to exsanguination (8.8%). It eliminated the severe type of external hemarthrosis. It did not reduce the incidence of internal hemarthrosis. It appears that, in order to completely eliminate the external and internal hemarthrosis, attention will have to be directed toward improving or eliminating the gambreling procedure.—[*R. L. Kitchell, W. J. Aunan, L. W. Murphy, and D. P. Mossberg: Shackling as a Factor in Ham Hip-Joint Hemorrhage. Am. J. Vet. Res., 18, (July, 1957): 491-501.*]

Fig. 3—Photograph of the coxofemoral joints of hams having internal hemorrhage (on the left) and external hemorrhage (on the right). The tear in the joint capsule is distended by a small piece of wood. Note the blood surrounding the joint on the right.

Salt Poisoning in Swine

The effects of increased intake of sodium chloride on swine were studied. Poisoning did not occur when water was given ad libitum. When water was restricted, for a time during the period of high sodium chloride intake, pruritus and constipation were evident and, after one to five days, many of the animals became blind and did not respond to external stimuli. Aimless wandering, forced circling, and epileptiform convulsions characterized later stages of the disease. The onset of a convulsion was signaled by twitching of the nose followed, in sequence, by clonus of the neck muscles, circling or running, retropulsion and, finally, salivation, rigidity, apnea, and cyanosis. Levels of sodium and chloride in the blood serum were increased; potassium and calcium levels remained within the normal range.

At necropsy, no specific gross lesions were apparent. Microscopic changes in the brain consisted of edema and meningoencephalitis, characterized by infiltration of eosinophils. Polioencephalomalacia, with proliferation of endothelial and glial cells, occurred later. The lesions were most prominent in the cerebral cortex.

The feeding of sodium propionate produced similar clinical and pathological manifestations.—[*D. L. T. Smith: Poisoning by Sodium Salt—A Cause of Eosinophilic Meningoencephalitis in Swine. Am. J. Vet. Res., 18, (Oct., 1957): 825-850.*]

The minimum supplemental sodium chloride requirement for cows producing 12,000 lb. of milk per year is about 1 oz. per head daily. Cows prefer loose salt but will consume all they need from block salt.—*Salt Inst., Chicago, Ill. (1957).*

Salt in Sheep Rations.—The addition of 0.25 per cent of sodium chloride to various sheep rations resulted in a growth response of 19 to 58 per cent over controls on the basal rations which contained 0.009 to 0.062 per cent of sodium, and 0.05 to 0.42 per cent of chlorine. Sodium bicarbonate supplement was as effective as sodium chloride. The sodium requirement of sheep was in excess of 0.06 per cent of the ration (0.88 Gm. per day). The digestibility of all components of the ration was reduced 3 to 4 per cent when salt was withheld. Addition of potassium bicarbonate created a circulatory deficiency of sodium and a severe dehydration. Farm animals seem to require salt to overcome the antagonistic effects of the high potassium content of animal feeds.—*Nutr. Rev. (Oct., 1957): 305.*

The Encephalitis of Hog Cholera.—Encephalitis is frequently a feature of hog cholera (swine fever) without the virus necessarily becoming neurotropic. Inflammatory lesions in the nervous system are probably caused by action of the virus on the autonomic nervous and endocrine systems.—*Vet. Bull. (Sept., 1957): Item 2691.*

Migratory Route of Hog Cholera.—When 17 shoats, in France, were injected subcutaneously in the left hock with 1 ml. of hog cholera virus, and were killed at intervals between postinoculation hours 4 to 72, there were no visible changes in the draining popliteal lymph nodes up to about 48 hours. In 1 pig, characteristic infarcts developed in the spleen in 20 hours. The pigs killed after 48 hours showed the characteristic septicemic lesions. The virus was present in the left popliteal lymph node in six hours, in the right node in 24 hours, and in the blood in eight hours.—*Vet. Bull. (Sept., 1957): Item 2692.*

The Leech as a Virus Reservoir.—Leeches fed on pigs with hog cholera contained virus for as long as 87 days and, in three instances, transmitted the disease to susceptible pigs while feeding on them. Those fed on rabbits with myxomatosis retained that virus for 154 days. When fed first on pigs with hog cholera and later on rabbits with myxomatosis, leeches acquired both viruses and retained the former for at least 122 days, the latter for at least 110 days. However, when fed first on the rabbits and later on the pigs, leeches acquired only the myxoma virus. The viruses were

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Correction—Hog Cholera in Georgia

In this item which appeared in the Oct. 15, 1957, JOURNAL, column 3 of table 1 shows the vaccine used in swine which later developed hog cholera. Dr. M. W. Hale advises that the abbreviation "MLV" used in the table refers to "modified live virus" vaccines in general. He was not sure that the specific trade-marked product "M.L.V." was involved in any of the reported cases.—Ed.

demonstrable in the gut contents of the leeches, but not in the body tissues, indicating that leeches are not biological carriers of these viruses, but harbor them in the gastrointestinal tract.—*R. E. Shope in J. Exptl. Med., 105, 373. Abstr. in Vet. Bull (Sept., 1957): Item 2712.*

New Drugs.—In 1956, 401 new drugs were made available to physicians—a cumulative total of 3,286 such in nine years. Most of these new drugs could be classified as laxatives, hypotensive or ataractic drugs, antispasmodics, antibiotics, antihistaminic agents, and hormones. Of the 3,286 new products, 333 were new single chemicals, 875 were duplicate products (single chemicals with different trade names), and 2,078 were compound products with more than one active ingredient. —*Ann. Allergy (May-June, 1957): 359.*

Efficiency of Tranquilizers.—Physicians warned, at a meeting of the British Medical Association, that the performance of tranquilizer drugs is much inferior to the claims made for them. In a survey of 8,200 patients, the benefits produced were small compared with the dangers. There had been four deaths, one in every five patients was made worse, and it was rare for a patient to feel better beyond the first week or so. Women who used tranquilizers for long periods gained much weight.—*J.Am.M.A. (Aug. 31, 1957): 2076.*

Infected Bone Meal.—Three clostridia sp, (*septicum, welchii,* and *chauvoei*) were recovered in culture and by guinea pig inoculation from samples of bone meal fertilizer in India. This was believed to be responsible for an increased instance of blackleg in the area.—*Vet. Bull. (Sept., 1957): Item 2628.*

Mucosal Disease.—Attention is called to the "Annual Report of the Veterinary Pathologist" (Kenya) for 1914-1915, in which a disease "C.H.," which resembled mild rinderpest, was described. The animals showed dullness, a high temperature for three or four days, a dark fetid diarrhea which later changed to constipation, a discharge from the eyes, and inflammation of the mouth, but no deaths from natural infection. The etiological agent was a virus bound to the red blood corpuscles. Rinderpest-immune cattle contracted the disease when inoculated, calves reacting more severely than adults, and Indian buffaloes more severely than calves.—*Vet. Rec. (Sept. 21, 1957): 916.*

Rinderpest Eradicated in Tanganyika

For the first time in 70 years, rinderpest was absent from Tanganyika for a full year, the last known case being in May, 1955. The disease was introduced from Egypt during the panzootic in East Africa in 1885. Organized control efforts, started after World War I, gradually pushed the infected area northward but major epizootics in 1934, 1947, and 1954 spread it southward. In 1937, control measures were abandoned in the northern areas and concentrated on stemming the southward advance; however, the established belts of immunized cattle were by-passed by infected game and, by 1939, rinderpest threatened to extend into North Rhodesia and Nyasaland. During 1939 and 1940, the countries to the south cooperated by furnishing personnel and funds to an organization which consisted of units for inoculation, propaganda, check, game research, and vaccine production. About 1 million cattle were vaccinated before the organization was disbanded two years later, after the infection had been controlled south of a rail line across central Tanganyika.

In 1947, the entire country was declared free of rinderpest but the disease was soon reintroduced from Kenya and, by 1949, it had again spread to the central railway lines, chiefly carried by wild animals. The next campaign was complicated by inadequate vaccine supply and, on one occasion, by contamination of a large consignment of vaccine with a strain of wild rinderpest virus. In 1953, the country was again declared free of known rinderpest but, again, it reappeared in a few months, this time where game could not be implicated.

Between 1940 and 1954, 24 million cattle were vaccinated; yet, in 1954, the disease appeared in 47 places and caused 6,462 deaths. In 1955, inoculation teams were organized on an area-responsibility basis, with greater attention to the inoculation and preservation of the fragile live vaccine, partly by expansion of refrigerating equipment. Efficiency reached its highest level in 1955 and 1956.—*Tanganyika Ann. Rep. for 1956, p. 1.*

Vasculature of External Nares and Related Areas in the Dog

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THE RECENT DEVELOPMENT[6] of a simple, practical procedure for surgical relief of stenotic nares in the dog has awakened interest in potential sources of hemorrhage from the nasal region. Although anatomists have previously described the principal arteries to the nose of the dog[1,5,7] and to the nasal septum of the deer,[10] the finer angio-architecture of the external nares of the dog has not been fully discussed. This current study is a by-product of comprehensive research concerned with coronary circulation in all domestic animals. By means of injection-corrosion techniques as well as conventional injection-dissection procedures, it has been possible to ascertain the detailed pattern of nasal vasculature in the dog.

MATERIALS AND METHODS

Two principal methods of study were used in this investigation. First, animals were embalmed and the blood vessels prepared for dissection by injection of neoprene resin. Second, vinyl acetate and neoprene resin solutions were injected into fresh specimens after perfusion with 1 per cent sodium citrate, according to procedures previously described by the senior author.[2,3] Vinyl acetate casts of the vascular organization of the external nares were produced by corrosion of specimens in 37 per cent hydrochloric acid.

ANATOMICAL FINDINGS

Directly or indirectly, the external nares of the dog receive their arterial blood through the lateral and dorsal nasal branches of the infra-orbital artery, the major palatine artery, the dorsal labial branch of the facial artery, the sphenopalatine, and ethmoidal arteries.

The facial artery, a branch of the external maxillary artery, supplies three superficial vessels to the head—the ventral labial artery to the lower lip and orbicularis oris muscle; the angularis oris artery to the angle of the mouth and anastomotic branches to the dorsal and ventral labial arteries; the dorsal labial artery (fig. 1) to the angularis oris and infra-orbital arteries and the upper lip.

The internal maxillary artery supplies the principal arterial vessels to the region of the external nares. As the terminal branch of the external carotid artery, the internal maxillary sends numerous branches to the mandible, temporal muscle, meninges, and orbit before being distributed to the face, walls of the nasal cavity, turbinates, and external nares. After the internal maxillary artery emerges from the alar canal, the external ophthalmic, pterygoid, anterior deep temporal, and buccal arteries leave it to supply their respective regions. The minor palatine artery is given off before the internal maxillary terminates in a common trunk for the infra-orbital, sphenopalatine, and major palatine arteries.

The minor palatine artery arises from the internal maxillary artery just posterior to the last molar tooth. It first curves anteriorly and then, at the junction of soft and hard palates, posteriorly to end at the free edge of the soft palate. In its tortuous course through the soft palate, the minor palatine artery gives off numerous branches to the pterygoid and palatine muscles, palatine glands, mucosa, and posterior part of the hard palate. It anastomoses with the minor palatine artery of the opposite side and with the major palatine artery.

The major palatine artery (fig. 2), the first branch of the terminal arborization of the internal maxillary, enters the palatine canal through the posterior palatine foramen. It gives off several small branches within the canal to the neighboring bone and emerges at the greater palatine foramen, opposite the fourth upper premolar tooth, to reach the ventral surface of the palatine portion of the maxilla and the palatine bone. It passes anteriorly through the erectile tissue of the hard palate, equidistant between the roots of the cheek teeth and the median plane.

Numerous small branches are distributed to the erectile tissue and mucosa. Many of these anastomose with branches from its fellow palatine artery. These also supply the lingual portions of the gums. Anteriorly, the major palatine artery divides into two principal branches at the level of the canine teeth (fig. 2). The first runs mesad to enter the palatine fissure where it curves dorsally to supply the maxilloturbinate (concha nasalis inferior), nasal septum, and mucosa of the nasal floor. The terminal branch of the major palatine continues toward the incisor teeth where it arches and anastomoses with its fellow. Branches from this also supply the lingual surface of the gums in addition to sending twigs between the canine and incisor teeth to anastomose with the lateral nasal branch of the infra-orbital artery.

The sphenopalatine artery (fig. 3, 4) passes through the sphenopalatine foramen to enter the

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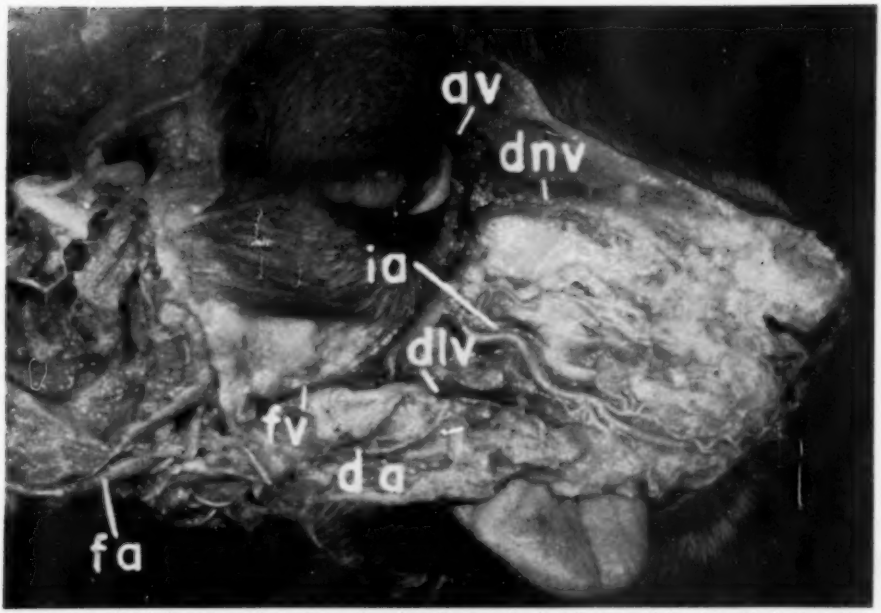

Fig. 1—Superficial dissection of the face of a dog showing the facial artery (fa), infra-orbital artery (ia), facial vein (fv), dorsal nasal vein (dnv), dorsal labial vein (dlv), angularis oculi vein (av), dorsal labial artery (dla).

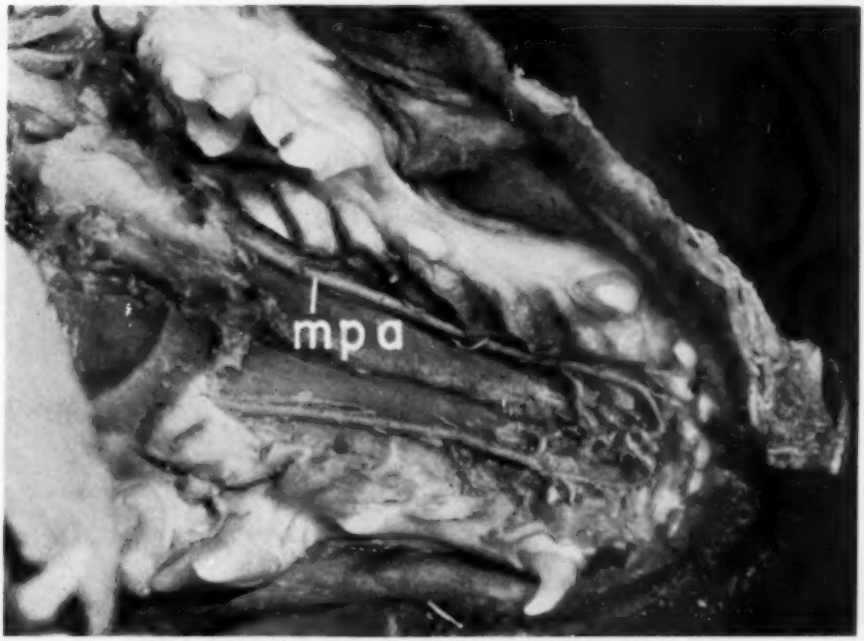

Fig. 2—Major palatine artery (mpa) of a dog showing terminal arches.

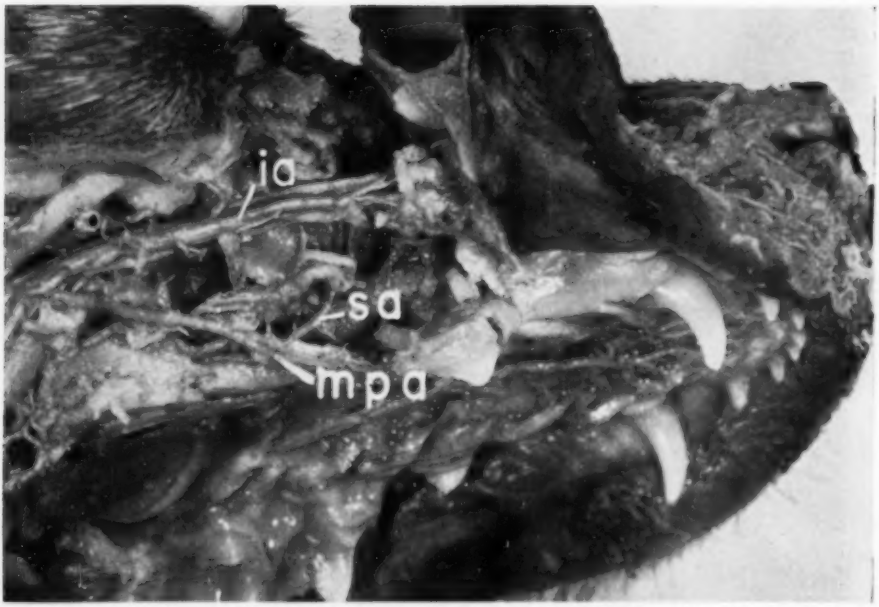

Fig. 3—Dissection of maxilla (sculptured) of a dog to illustrate the relationships of the following arteries: infra-orbital (ia), sphenopalatine (sa), major palatine (mpa).

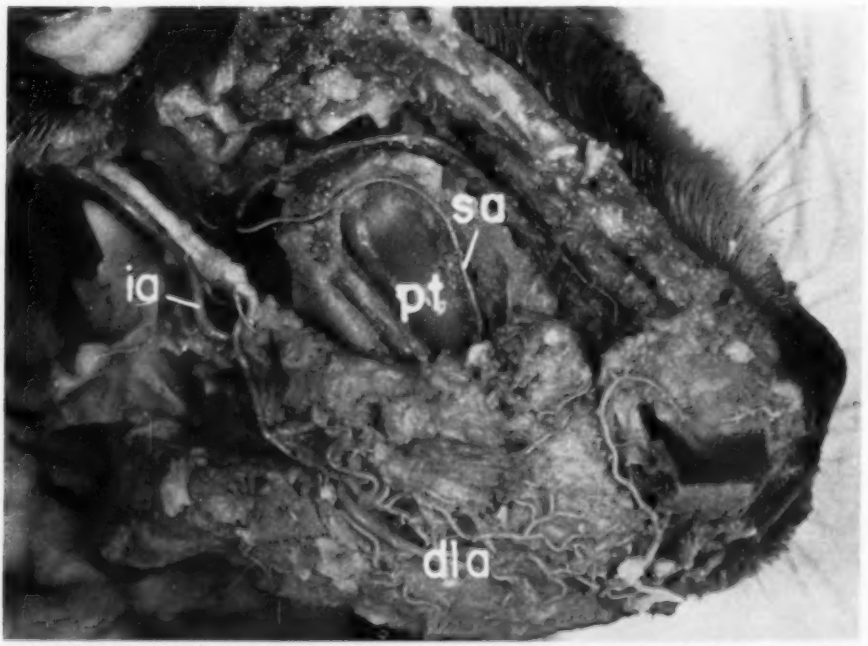

Fig. 4—Partial removal of maxillary and nasal bones to show the following arteries of a dog: sphenopalatine (sa), dorsal labial (dla), infra-orbital (ia). A permanent canine tooth (pt) is present.

nasal cavity. It traverses the ventrolateral surface of the maxilloturbinate, curves dorsad at the level of the infra-orbital foramen to the nasoturbinate (concha nasalis superior), and runs anteriorly along the latter to ramify under the alar cartilage of the nostrils. Branches of the sphenopalatine artery are also distributed to the ethmoturbinate (concha nasalis media) and to the mucosa of the walls, floor, and septum of the nasal cavity.

The infra-orbital artery (fig. 1, 3-5) enters the maxillary foramen to traverse the infra-orbital canal. Its first branch, the malar artery, leaves the dorsal surface of the parent trunk just before it reaches the maxillary foramen. The malar artery sends a nutrient branch into the lacrimal bone posteromedial to the posterior opening of the nasolacrimal duct and also to the osseous lacrimal duct. The main trunk of the malar artery continues to the lower eyelid where it terminates in numerous small branches. Anastomoses with the superficial temporal and facial arteries are present.

The infra-orbital artery gives off small, evenly spaced, dorsal alveolar arteries in its course through the infra-orbital canal. These supply the roots of the upper cheek teeth and the neighboring bone.

At the infra-orbital foramen, located at the approximate center of the maxillary surface, the infra-orbital artery emerges and divides into dorsal and lateral nasal arteries. The dorsal nasal artery, the smaller of the two branches in diameter, runs forward to the dorsum of the nose and the upper lip. It anastomoses principally with the lateral nasal artery. The lateral nasal artery sends numerous fine branches to the ventral portion of the upper lip and adjacent gums. One large branch ascends around the lateral edge of the external naris (fig. 4-6). The terminal branch ascends along the philtrum and anastomoses with its fellow on the midline as well as with the major palatine, dorsal nasal, and dorsal labial (facial) arteries.

Finally, the external nares receive arterial blood from branches of the anterior cerebral arteries. Unilaterally, an artery leaves the anterior cerebral artery, anterior to its junction with the middle cerebral artery. It does not follow the optic nerve, as does the internal ophthalmic artery,[1,2] but runs forward along the floor of the cranial cavity to the

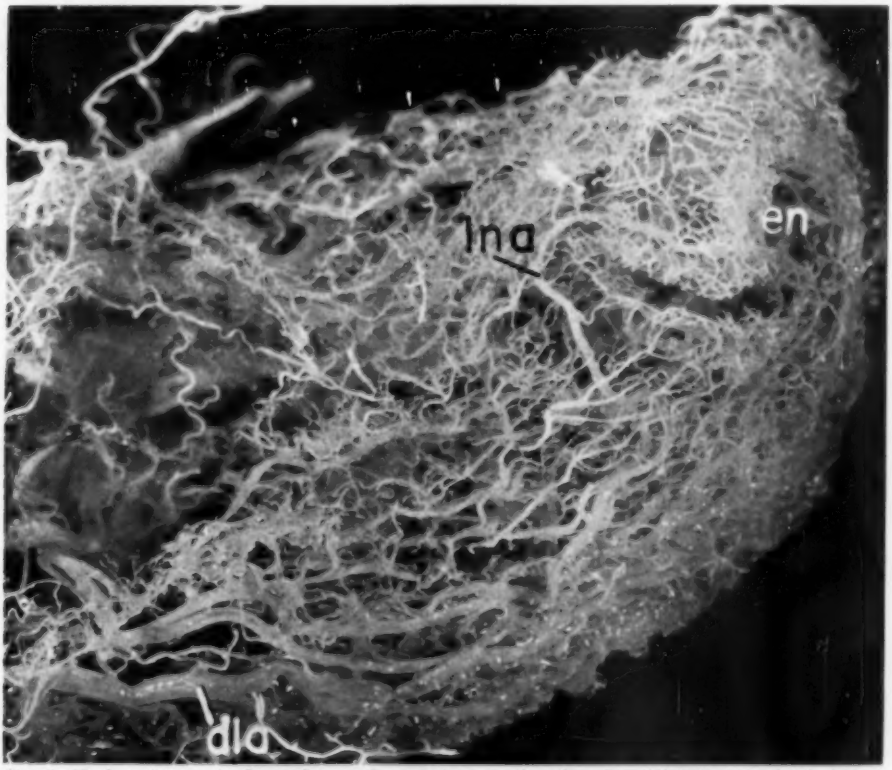

Fig. 5—Vinyl acetate cast of vasculature of external nares of a dog. Dorsal labial artery (dla), lateral nasal artery (lna), external nares (en).

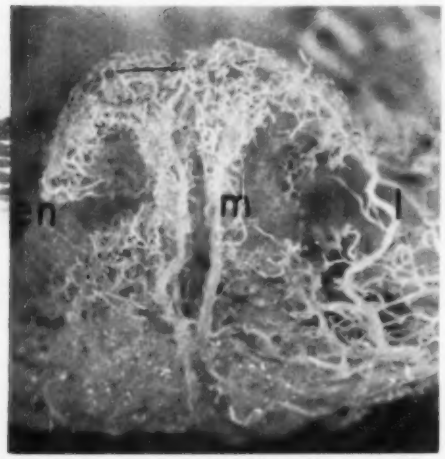

Fig. 6—Anterior view of vinyl acetate cast of external nares of a dog. External nares (en), medial branch of lateral nasal artery (m), lateral branch (l).

Fig. 7—Anterodorsal view of vinyl acetate cast of the nose showing complex pattern of arteries and veins in a dog.

dorsal edge of the cribriform plate. The artery enters the nasal cavity at this point and divides into three branches. The uppermost branch runs in the nasal mucosa along the junction of the nasal septum and nasal bone, passes under the alar cartilages, and finally terminates superficially in the external nares. The lower two branches are distributed to the mucosa of the nasal septum and ethmoturbinate. They anastomose freely with the previously described uppermost branch and with branches of the sphenopalatine artery. The distribution of this artery parallels the course of the anterior ethmoidal artery of man[6] in that it is distributed to the middle and anterior parts of the nose and to the skin around the external nares. In the dog, the vessel arises from the anterior cerebral artery rather than from the ophthalmic artery as in man. Although the ethmoidal artery of the dog does supply the ethmoturbinates, it is sometimes described as being a terminal branch of the anterior cerebral artery, not extending as far forward as the external nares.[7] It has also been described as being a branch of the external ophthalmic artery.[7,9] For lack of a better name, the artery found in our specimens will also be designated as ethmoidal. However, its origin is not consistent

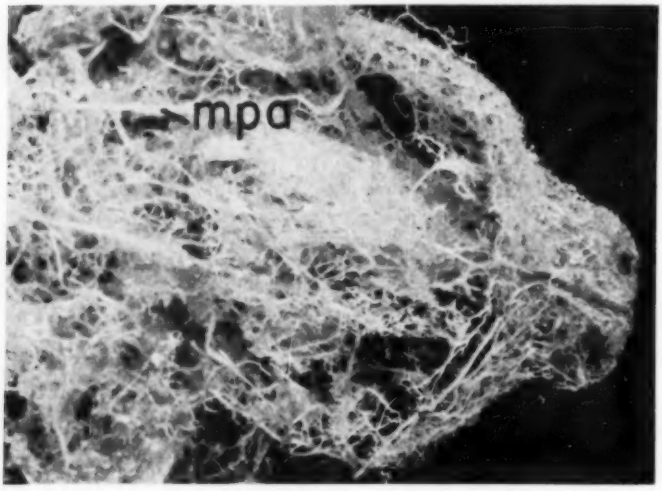

Fig. 8—Vinyl acetate cast of vessels of the nose of a dog illustrating vasculature of upper lip and nasal septum (ventral view). Major palatine artery (mpa).

with that of a classically described ethmoidal artery and its distribution is more generalized.

The deep veins of the external nares and related areas are, for the most part, satellites of the arteries. Superficially, the principal drainage of the external nares is via the various tributaries of the facial and diploic veins. The facial vein is formed from the union of the dorsal nasal and angularis oculi veins in the lateral nasal region (fig. 1). In the area of the infra-orbital foramen, it receives lateral nasal and dorsal labial veins. At the ventral border of the mandible, after acquiring veins from intermediate areas, the facial vein continues posteriorly as the external maxillary vein. The region of the external nares contains rich plexuses of arteries, veins, and capillaries (fig. 5-8).

DISCUSSION

Recent interest in the surgical correction of stenotic nares in the dog has led to a detailed study of the vascular anatomy of the external nares. The complexity of vessels is described and illustrated in order to emphasize the fact that, although numerous arteries and veins contribute to the angio-architecture of this region, forming a delicate, lacelike pattern, no vessel or appreciable size is located in this particular site. As pointed out,[6] the tissue in this area is vascular and some hemorrhage is encountered during the operation. Multitudinous small arteries, veins, and capillaries contribute to the high degree of vascularity in this region. Because of the varied, numerous sources of blood to the region of the external nares, and also because of the complex anastomoses, traumatic or surgical injury to any one area of the head or face will not result in deprivation of blood to the external nares. Conversely, because of the intricacy and fineness of the vessels, surgical incision inevitably results in generalized hemorrhage from the cut surfaces, but does not encroach upon major arteries.

SUMMARY

A study of the surgical anatomy of the external nares and related areas, with emphasis upon major sources of blood and the vascular complexity of the region, reveals that the external nares are surrounded by an intricate pattern of arteries, veins, and capillaries. Arterial blood reaches the area through detailed anastomoses among the following vessels: dorsal labial artery (branch of facial artery); dorsal and lateral nasal arteries (branches of the infra-orbital artery); sphenopalatine artery; major palatine artery; and the ethmoidal artery (branch of the anterior cerebral artery).

Venous drainage of the region is by means of deep satellite veins of the arteries as well as by superficial tributaries of the facial vein.

Local injury to any part of the head will not deprive the external nares of circulation. Conversely, an incision of the nares will involve no major arteries.

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Cat Scratch Disease.—Six cases of cat scratch disease, 4 in children and 2 in adults, are reported in the *British Medical Journal* (Aug. 24, 1957, pp. 444 and 446). In one adult, a typical syndrome was complicated with pneumonia which could be assigned to no other cause. All were positive to intradermal tests with cat scratch antigen. In four persons, the only lesion was an enlarged or suppurative lymph node—in the axilla of two and in the groin of two. One girl had an inflamed right eye and enlarged lymph nodes around her right ear. The woman with pneumonic complications had a lesion on the palm of her hand and enlarged lymph nodes at the elbow and axillary space. All had been in contact with cats, none of which showed signs of illness.

The average age of farmers is on the increase. In 1910, it was 41.9 years, now it is 49.3 years.—*Feed Bag (July, 1957): 101.*

Surgery and Obstetrics
and Problems of Breeding

A Preliminary Report on the Treatment of Cystic Ovaries in Dairy Cattle by the Injection of Gonadotropic Hormones Directly into the Follicular Cyst

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Ithaca, New York

ALL DAIRY PRACTITIONERS questioned by the author have substantiated his published data[5] that the incidence of cystic ovaries is increasing. This may be due to improved breeding practices, such as artificial insemination, and to improved management and feeding practices that have greatly increased the production of the average dairy cow. Cystic ovaries have been reported most frequently in the high-producing cows.[2,4] However, the hereditary nature of this condition has been observed by practitioners and has been reported by the above authors. A further study of daughters of bulls used in artificial insemination studs might give valuable information on the sire's role in the hereditary pattern of this disease. The increased incidence of cystic ovaries has caused serious breeding problems in some herds. The relatively high cost of the luteinizing hormones has placed an added burden on all progressive farmers who own good-producing cows so affected.

Although the condition of cystic ovaries or nymphomania in dairy cattle has been recognized by veterinarians for a long time, progress toward an understanding of the etiological factors had been slow until recent years. In 1944, it was reported[1] that an unfractionated anterior pituitary extract rich in the luteinizing hormone (LH) was highly effective in correcting cystic ovaries in cattle. This hormone, found in high concentration in pregnant women's urine (chorionic gonadotropin) as well as in certain pituitary preparations, has been found useful in the therapy of cystic ovaries.[3,8] Following the intravenous or intramuscular injection of LH into affected cattle, whether the cysts are ruptured or not, luteinization of the cystic follicles occurs and is palpable on rectal examination of the ovaries within several days in animals in which treatment is successful. The LH probably acts upon the granulosa cells and theca interna of the normal or cystic follicle producing lutein tissue necessary for a normal estrous cycle.

In 1955, it was demonstrated[5] that cystic ovaries in cattle are basically due to a derangement of the mechanism of ovulation and corpus luteum formation. In affected cattle, the release of LH from the basophilic *delta* cells of the anterior pituitary gland, which occurs in normal cattle early in estrus, fails or is retarded so that ovulation does not occur and the mature follicle or secondary follicles develop into ovarian cysts. The reason for this failure of release of LH from the anterior pituitary was not determined. It is suspected that the hypothalamus probably has some influence on the anterior pituitary gland causing the release of LH at the proper time. There was no apparent lack of LH in the anterior pituitary gland in cows with cystic ovaries when based upon the appearance of the gonadotropic cells or assays.[5,6] That the bovine cystic ovary is an anomaly of ovulation is further substantiated clinically by the history of cows which have normal estrous cycles, followed by nymphomania or signs of continuous estrus immediately after the last apparently normal estrus. The author has observed a number of cows, that have had cystic ovaries and nymphomania develop following estrus, recover after treatment with LH only to have the condition recur after the next normal estrus. After re-treatment, recovery and conception occurred when a small dose of LH was given during the subsequent estrus.

The number of valuable purebred cows that fail to respond to repeated treatments, even with large doses of the luteinizing hormones, has increased in recent years. It has been reported[10] that cows injected repeatedly with the gonadotropic hormone develop a refractoriness to it. This is possibly due to antihormones produced by the animal, since this hormone is a protein.

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Better results were reported[8] in relapsing cases of cystic ovaries if they were retreated with a luteinizing hormone from a different source than the one originally used.

In 1953, a report was made[7] on 80 cows with cystic ovaries treated by the injection of 250 to 1,000 I.U. of chorionic gonadotropin in 5 cc. of distilled water through the vaginal wall into the follicular cyst. Of the 80 cows, 52 (65%) recovered, 6 after the second injection. Of these 52 cows given a total of 59 services, 45 per cent became pregnant. In 1957,[9] of 56 nymphomaniac cows treated similarly with 750 to 1,250 I.U. of chorionic gonadotropin in 5 cc. of distilled water, 36 recovered after one injection and 8 of 11 after two injections, a recovery rate of 78.4 per cent. Of the 44 recovered cows, 40 (71.4%) conceived, 34 after one service. These trials demonstrated that fairly good results, with no harmful effects, could be obtained by the injection of LH directly into the follicular cyst.

This method of treatment of cows with cystic ovaries utilizes small doses of LH which reduces the cost of treatment; and it introduces the luteinizing hormone directly into the cyst to be luteinized, thus avoiding the possibility of inactivation of the hormone by antihormones in the body fluids such as may occur if they are given intravenously or intramuscularly. A further investigation of this form of therapy for cows with cystic ovaries is here reported.

METHODS

The technique reported[7,9] for injecting LH into follicular cysts consisted of introducing a small gauge needle attached to a rubber tube into the vagina with one hand, passing the needle through the vaginal wall dorsal or dorsolateral to the cervix, and using the other hand in the rectum to direct the cystic ovary onto the needle. After some of the cystic fluid was drained off, the luteinizing hormone in 5 cc. of distilled water was injected. In most cases, this caused a filling and distention of the cyst that was palpable by the fingers holding the ovary. The above authors recommended administering a light epidural anesthesia before this operation. However, this was found to be unnecessary. There are no sensory nerves in the anterior portion of the vagina or in the ovary and, in some cases, ballooning of the rectum accompanied the anesthesia.

After a number of trials with different procedures, the following two techniques were found most satisfactory. One of these was essentially the same as described above except that 1 to 2 cc. of fluid was injected, since 5 cc. caused some of the smaller cysts to rupture. When the smaller volumes were injected, the large rubber tube was unsatisfactory; so a polyethylene tube 14 inches long and 0.045 inches in inside diameter with couplers* was attached to a 2-inch, 18-gauge needle and a 5-cc. glass syringe. To prevent rupture of the cyst, some fluid had to be removed. Using a syringe to draw fluid from the cyst and then detaching this syringe and injecting the hormone was not as satisfactory as allowing a few cubic centimeters of cystic fluid to drain through the tube. The cystic fluid could be removed so rapidly with a syringe that the cyst would collapse and, in some instances, by the time the syringe was detached and the hormone was injected, the point of the needle had become disengaged from the collapsed cyst cavity. While the vaginal technique could not be used in heifers, it is preferred for pluriparous cows, especially those of the large dairy breeds with genital organs and ovaries that had dropped forward and downward into the peritoneal cavity.

The second technique is indicated because in most cattle there is less danger of introducing infection, it is simple, and it requires little equipment. It consists of injecting the hormone into the follicular cyst through a 4½-inch, 18-gauge needle inserted through the ischiorectal fossa. A small skin area in the anterior portion of the left or right fossa, just posterior to the sacrosciatic ligament and lateral to the coccygeal vertebrae, is shaved with a Bard Parker blade and disinfected. In larger cows, the side prepared depends upon which ovary is cystic; in smaller cows, either ovary may be injected from either fossa. The tail is pushed forward and upward for restraint as the needle is inserted through the skin. Then the arm is inserted into the rectum and the cystic ovary is brought dorsally and posteriorly as the needle is moved anteriorly and ventrally. When the needle penetrates the cyst, the follicular fluid spurts out. Depending on the size of the cyst, after about 1 to 4 cc. of fluid has escaped, the luteinizing hormone in 1 to 2 cc. of diluent is injected into the cyst and the needle is promptly withdrawn. This technique is necessary for injecting cysts in heifers and is preferred by the author for injecting cysts in young cows and cows of the smaller breeds. In many cases when the history is indicative and the external signs of cystic ovaries are present, the needle is inserted through the skin even before the ovaries are examined. Thus the examination and treatment can be completed in one operation.

Of 22 cows treated for cystic ovaries, 4 were treated twice and 1 cow three times — a total of 28 injections. These were unselected cases in purebred, commercial, and institutional herds. The 22 cows had an interval from the last calving to the time of treatment ranging from 30 to 300 days with an average of 151 days. The duration of signs of nymphomania shown by 19 of the 22 cows ranged from 2 to 270 days; an average of 56.2

*Clay Adams Co., Inc., New York, N. Y.

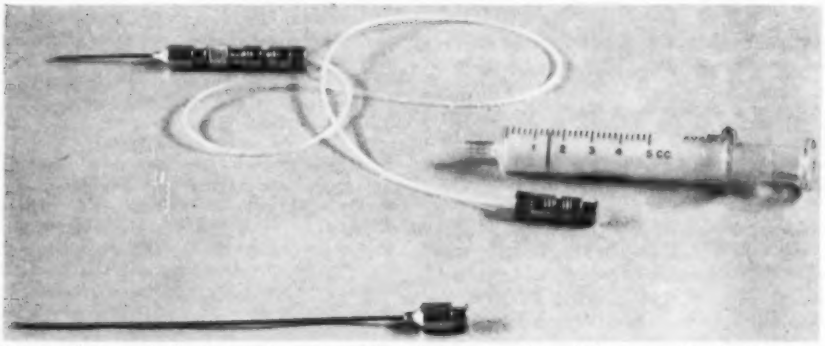

Fig. 1—Photograph showing equipment used for injecting gonadotropic hormones directly into ovarian cysts of cattle.

days. The other 3 cows showed signs of anestrus, the duration of which was not known. In these preliminary studies, 1 to 3 cc. of diluent containing from 250 to 2,500 I.U. of chorionic gonadotropin (Follutein*) was used on 17 cows; 9 cows were treated with 0.5 to 5.0 rat units (r.u.) of sheep pituitary gonadotropin (Vetrophin†), and 2 cows were given 25 and 50 Armour units (A. U.), respectively, of pituitary gonadotropin PLH.**

Most of these cows were examined from two to ten days later to determine the rate and degree of the luteinization of the cystic follicle. The clinical findings, procedures followed, and results of therapy were carefully recorded.

RESULTS AND DISCUSSION

In this preliminary report of work carried out during the last ten months, only 11 of the 22 cows treated for cystic ovaries have as yet been found pregnant, 3 have had regular cycles but have not been rebred, 3 have been bred two to four times, 3 have been bred two times at regular estrus and sold because they failed to conceive, 1 cow again developed cystic ovaries after one or two normal cycles, and 1 cow failed to recover. Recovery was denoted by cessation of the signs of nymphomania or anestrus, formation of a corpus luteum, as determined by rectal examinations, and occurrence of a normal estrus. In 2 cases, relapses occurred after one or more estrous cycles but this can not be ascribed to a failure of the treatment.

Of 17 cows treated by injecting 250 to 2,500 I.U. of chorionic gonadotropin into the follicular cyst, 13 (76.4%) recovered; 4 did not recover. Of 3 cows treated with 250 I.U., 1 recovered and 2 did not; of 7 cows treated with 500 I.U., 5 recovered and 2 did not; of 5 cows treated with 1,000 I.U., all 5 recovered; and of 2 cows treated with 2,000 and 2,500 I.U., respectively, both recovered. The dose of 250 I.U. of chorionic gonadotropin may be too low to be considered effective and practical. If the 3 cows so treated are removed from the totals, 12 (85.7%) of the remaining 14 cows treated with 500 to 2,500 I.U. of chorionic gonadotropin recovered. Of 12 cows treated with 500 to 1,000 I.U., 10 (83.3%) recovered.

Of 9 cows treated by injecting 0.5 to 5.0 r.u. of sheep pituitary gonadotropin into the follicular cyst, 7 (77.7%) recovered and 2 did not recover; of 2 cows treated with 0.5 r.u., both recovered; of 5 cows treated with 2.0 to 2.5 r.u., 3 recovered and 2 did not; of 2 cows treated with 3 and 5 r.u., respectively, both recovered.

Two cows treated by injecting 25 and 50 A.U. of pituitary gonadotropin into the follicular cyst recovered.

Of 28 cases of cystic ovaries treated by the above doses and types of luteinizing hormones injected into the follicular cyst, 22 (78.6%) recovered. This compares favorably with the results the author reported previously,[8] in which an 83.5 per cent recovery rate in 127 cows treated with 1,000 to 5,000 I.U. of chorionic gonadotropin intravenously and an 84.9 per cent recovery rate in 146 cows treated with 10 r.u. of sheep pituitary gonadotropin was recorded.

Five of the 22 cows treated for cystic ovaries by the intrafollicular injection of a gonadotropic hormone had been previously

*Follutein—Squibb and Sons, Brooklyn, N. Y. †Vetrophin—Abbott Laboratories, North Chicago, Ill. **PLH—Armour Veterinary Laboratories, Kankakee, Ill.

treated one to four times unsuccessfully by the intravenous or intramuscular injection of the recommended doses of commercially available gonadotropins. All 5 cows recovered. Two recovered after the first intracystic injection of 2,000 I.U. of chorionic gonadotropin and 5 r.u. of sheep pituitary gonadotropin, respectively. Two cows recovered after the second treatment with 25 A.U. pituitary gonadotropin and 500 I.U. chorionic gonadotropin, respectively, after the first treatment with 2.5 r.u. sheep pituitary gonadotropin and 250 I.U. chorionic gonadotropin, respectively, had failed. Of these 4 cows, 1 is pregnant, 1 recovered but has not been bred, and 2 recovered and were bred several times but were sold since they failed to conceive. The fifth cow recovered after the third treatment with 50 A.U. pituitary gonadotropin after 250 I.U. chorionic gonadotropin and 2.5 r.u. sheep pituitary gonadotropin had failed, but relapsed after one or two normal heat cycles.

Repeated rectal examinations on treated cows revealed no evidence of adhesions or other damage to the ovary or surrounding tissues. Two cows with cystic ovaries had their ovaries "tapped" three or four times with an 18-gauge needle before slaughter at a local packing plant. With the exception of a slight blood-tinging of the cystic fluid in one cyst, no damage was observed by the repeated puncturing of the ovaries.

From this preliminary trial, it appears that the injection of relatively small doses of luteinizing hormone directly into the follicular cyst is a procedure worthy of further study. It is possible that small doses of a gonadotropic hormone, such as 500 or 1,000 I.U. of chorionic gonadotropin, given intravenously might produce similar results to those reported for the intrafollicular injection of the hormone. The author hopes that this report will stimulate further study and observations on this mode of therapy, especially as to its value in those refractory cases that fail to respond to the intravenous or intramuscular injections of the luteinizing hormone.

SUMMARY

1) Two techniques for the injection of luteinizing hormone into cystic follicles are described, *i.e.*, the vaginal approach and the insertion of a long needle through the ischiorectal fossa.

2) There was no clinical or postmortem evidence of any significant damage or injury produced by tapping cystic ovaries with a fine gauge needle and injecting a gonadotropic hormone.

3) Preliminary results indicate that this mode of therapy for cows with cystic ovaries may be practical since small doses of gonadotropic hormones, when injected into the cystic follicles, produced a satisfactory recovery rate.

4) Encouraging results with this mode of therapy have been observed in a limited number of cows with cystic ovaries that failed to respond to the intravenous or intramuscular injection of a luteinizing hormone.

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Ovarian Inactivity in Related Heifers.—In 6 Ayrshire heifers, 18 to 24 months old, from the same sire, estrus had not occurred in 3 and the other 3 had not conceived. All 6 had infantile uteri and abnormally small vulvas, and corpora lutea were found only in one ovary of each of the latter 3. Injection of 30 mg. of stilbestrol in the 3 older heifers had no effect. Later, all 6 were given 2,000 I.U. of follicle-

stimulating hormone (F.S.H.) intramuscularly and, in about 48 hours, all showed estrus. Because of possible superovulation, they were not mated until the following estrus. Two conceived at that time and all conceived in the next few months. The season as well as heredity may have influenced this delay in maturing.—*N. R. Gordon in Austral. Vet. J. (June, 1957): 151.*

Dysfunction of the Bovine Ovary.—Most ovarian dysfunction in the cow is secondary to: (1) inadequate lutein hormone from the anterior pituitary gland, resulting in cystic degeneration of the follicles, cystic corpora lutea, and failure of or delayed ovulation; (2) pus, a mummified fetus, or a degenerating embryo in the uterus, causing persistence of the corpus luteum; and (3) malnutrition.—*S. J. Roberts in Vet. Rec. (July 13, 1957): 667.*

Mummified Fetuses in Cows.—In a period of 30 years, in a herd of Holstein-Friesian cattle, 29 of 2,607 cows produced 32 mummified fetuses (1.1% of cows, 0.43% of parturitions). Abortions occurred in 13 cows in less than seven months, and 4 cows carried the mummy over nine months (maximum, 342 days; mean, 215 days). None of these cows showed evidence of postparturient abnormalities except 5 which showed infection and 1 each with cystic ovaries and a retained corpus luteum. The abortion rate for these cows in other pregnancies was 6.0 per cent compared with 8.6 per cent for all cows.—*J. Dai. Sci. (Sept., 1957): 1030.*

Determination of Pregnancy in Cows.—Pregnancy can be determined with precision by rectal palpation, as early as day 30. Wearing a rubber sleeve protects the cow as well as the arm. Many who are right-handed prefer to use the left hand. The same hand should always be used as it can be "educated" to palpate much more accurately than the untrained hand.

At 33 days, a pea-sized (⅜ in.), fairly firm embryonic mass can be felt to slip between the fingers, usually in the mid-third of the horn, and a corpus luteum will be present, usually in the ovary of the same side. In twin pregnancies, an embryo and a corpus luteum may be present on each side, or two of both may be on one side. At 50 days, the mass will feel oblong, more soft, about 2 inches in diameter, and probably will contain 200 cc. of fluid. If membranes can not be palpated, some pathological condition may be present.

At 90 to 100 days, the fetus will be 4 to 7 inches long, and approximately a quart of fluid will be present. Later, the cotyledons and the middle uterine artery should be distinctly palpable. One cow, 65 days after she was bred, had no corpora lutea but the uterus was typical of a 45-day pregnancy. She aborted two weeks later; the fluids probably were being absorbed at the time of the examination. In about 50 per cent of cows in which the corpus luteum and the fetus were on opposite sides, abortion occurred.—*E. Woelffer in Vet. Sci. News (Summer, 1957): 11.*

Treating Nymphomaniac Cows.—Of 56 nymphomaniac cows, 36 recovered after one injection of chorionic gonadotrophin into the cysts and 8 of 11 others recovered after a second treatment. Estrus usually occurred in 18 to 22 days and 40 of the cows became pregnant, 34 at the first service.—*Vet. Bull. (July, 1957): Item 2262.*

Pericardiectomy in a Cow.—A young cow had been ill for several weeks with signs of cardiac failure. She had a fair appetite, was losing weight, and her pulse was weak, irregular, and 90 per minute. The jugular veins were distended, with pulsation visible 6 inches up the neck. The sternal area was edematous. Upon auscultation, there was a murmur-like sound related to the closure of the cardiac valve, and electrocardiographic examination showed slight auricular fibrillation. Except for a slight anemia, no other marked abnormalities were found. In the next two weeks, while allowed only 2 gallons of water daily, the edema and jugular pulse disappeared but the heart condition remained, and she lost 130 lb. Thoracotomy was then performed using an intravenous anesthetic and forced respiration. The distal 8 inches of the left fifth rib was removed, leaving the medial periosteum which was then incised. There was no excessive fluid in the cavities, the pleura and lungs appeared normal, but when the pericardium was open, a few fibrous adhesions to the epicardium were found, suggesting a previous acute pericarditis. As much of

the pericardium as possible was removed with scissors and the wound was sutured. The cow made a remarkable recovery but, having aborted and not milking, she was slaughtered. The heart appeared to be normal.—*C. Jennings and W. I. M. McIntyre in Vet. Rec. (Sept. 28, 1957): 928.*

Ova Transfer in Ewes.—The best ewe in a Suffolk flock, in Britain, and 7 other ewes with synchronizing estrous cycles were used. The donor ewe, 13 days after estrus, was given 900 I.U. of whole pregnant mare serum (PMS) subcutaneously and was bred during estrus, three days later. Sixty hours after onset of estrus, upon laparotomy, the ovaries showed evidence of five ovulations, and four ova were recovered from the fallopian tubes by flushing them with homologous serum. Two of the ova were damaged; the other two were placed in the uterus of another ewe, resulting in 1 normal lamb.

Ten days after laparotomy, the donor ewe was given 1,000 I.U. of PMS and was bred three days later. Sixty hours after mating, there was evidence of 14 ovulations and 14 ova were recovered. One was damaged but 13 were placed in the uteri of 6 other ewes, resulting in 9 normal lambs. In a third attempt, no ova were recovered so the donor ewe was placed with a fertile ram and later had 1 lamb normally. Thus, 11 lambs were produced from 1 ewe in one season. As the gonadotrophin treatments were repeated, there was evidence of decreasing ovarian response; longer intervals between treatments might bring better results.—*Rowson and Adams in Vet. Rec. (Sept. 7, 1957)*: *849.*

Possible Sex Control in Cattle.—Significant sex control in rabbits has resulted when using semen through which an electric current was passed, with spermatozoa being attracted to the opposite poles, a process called electrophoresis. Sperm which migrated to the anode usually produced females. In 167 trials with rabbits, the desired sex was obtained in 68 per cent of the offspring. In 7 of 31 litters, all of the young were of one sex. It has been reported in cattle and mice that males with low blood pH (acidity) sired mostly females; those with a high pH, mostly males. Research continues.—*H. A. Herman in Hoard's Dairyman (Sept. 25, 1957)*: *907.*

Mid-Dorsal Dermoid Sinuses in Rhodesian Ridgeback Dogs—A Case Report

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Rhodesian Ridgebacks have recently been recognized as a new breed by the American Kennel Club. With the introduction of this breed to the North American continent, the small animal practitioner should be alert for a condition that is peculiar to the breed—the development of a "mid-dorsal" cyst or "dermoid sinus." This is a congenital malformation, which appears most frequently over the cervical vertebrae and, to a lesser extent, over the sacrococcygeal region. Its development is ascribed to incomplete dehiscence between the spinal cord and skin just after the closure of the neural tube in early embryonic life. The frequency with which it appears indicates that inheritance is not due to a simple recessive factor, but to a gene complex; therefore, it is probable that most Rhodesian Ridgebacks carry some of the factors, and thus the condition could appear in any breed line.

The sinus can be felt as a thin cord, 1 to 5 mm. in diameter, when a deep fold of skin is grasped and the subcutaneous tissues allowed to slip back and forth between the fingers. When the area over the sinus is shaved, a minute opening with a tuft of hair protruding can be seen. Microscopically, the sinus appears to be an invagination of the skin. The epithelium is thin, the cornified layer consisting of one or two layers of cells; the hair follicles are shallow and partially functional; and the sweat and sebaceous glands are in a normal position in relation to other epidermal structures. The sinus extends, and is attached, to either the dura mater in the coccygeal region or the spinous process of a vertebra in the cervical region (usually the second cervical vertebra). In uncomplicated cases, it is filled with inspissated sebum, exfolia-

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ted skin debris, and hair; but these cases are usually not seen. The practitioner will see cases in which bacteria have contaminated the material in the sinus and produced inflammatory reactions with sequelae ranging from a mild myelitis to an acute encephalitis.

The term "dermoid sinus" is used in South African literature for describing the sinus, and seems most appropriate, even though texts on pathology and Dorland's *Medical Dictionary* have no such terminology except to refer to "dermoids" as "inclusions of dermal tissue along the lines of embryonic fissure."[1] The term "pilonidal sinus" would also seem correct because of the histological similarity of the sinus in Ridgebacks and the postanal dimple over the last coccygeal vertebra in man. However, they appear to differ since, in man, the sinus usually contains a hair or hairs with the apex pointing inward, indicating that the hair shaft may have been forced in by friction of clothing, but no such hairs have been reported in Ridgebacks. However, the possibility of hairs causing the sinus in Ridgebacks exists and sinuses should be carefully examined when removed. It has been suggested[4] that the pilonidal sinus in man is not truly congenital, but due to a combination of factors including body build and presence of the postanal dimple, causing an invagination of epithelium and the development of a sinus as a sequel to trauma by broken hairs; also, that there is a marked age incidence (18 to 27 years) in the development of the discomforting symptoms associated with the condition in man, which is at variance with the incidence of congenital lesions. On the other hand, complications of the sinus in Rhodesian Ridgebacks appear early in life and conform to the congenital pattern. It would, therefore, seem that the term "dermoid sinus" is the more appropriate one.

Treatment of the condition is radical excision of the tract.

CASE REPORT

A male Rhodesian Ridgeback, 5 months old, was presented at the clinic of one of the authors (J. G.) because of weakness in its hindquarters. There was posterior paresis, hyperesthesia of the sacrococcygeal region, and an elevated temperature. A palpable cordlike structure was found in the midline, dorsal to the third coccygeal

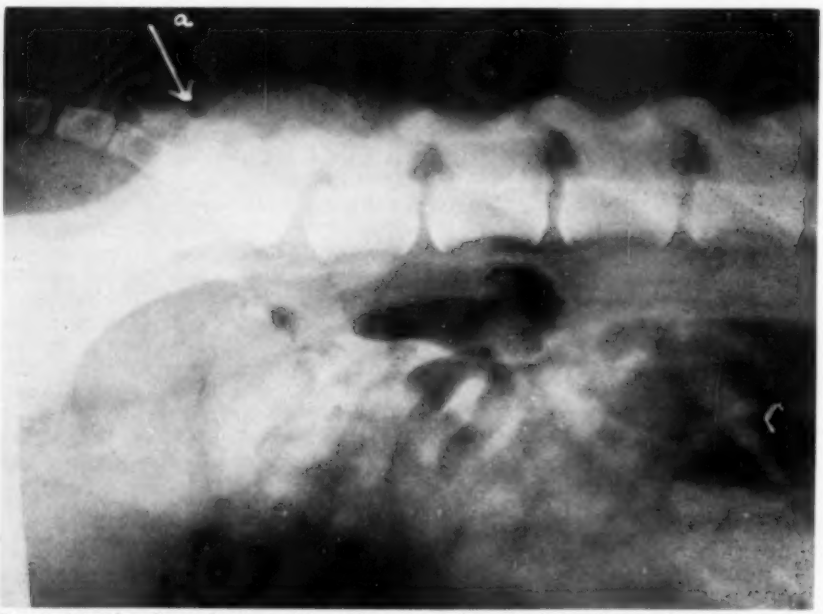

Fig. 1—Lateral radiograph of caudal vertebrae in a dog, showing evidence of an inflammatory process (a) at the junction of the last sacral and first coccygeal vertebrae (apophysitis).

vertebra, extending from the skin in a cranioventral direction toward the spine, and ending at the interspace between the last sacral and first coccygeal vertebrae.

The animal was referred to the clinic of the Ontario Veterinary College on July 16, 1956. The dog appeared alert and responsive and had a good appetite. The previous findings were confirmed; the temperature was 103.8 F. When the hair was clipped from the region, it was seen that the palpable cord came to the surface of the skin as a sinus with a 1-mm. opening from which short dark hairs protruded. After probing with a sterile, blunt needle, small drops of pus were expressed and collected for bacteriological culture and determination of antibiotic sensitivity.

Before determining the extent of the sinus by injecting a radiopaque dye, scout films of the area were taken. These showed the existence of inflammatory changes in apophyses on the last sacral and first coccygeal vertebrae, which indicated some connection between the sinus and this area (fig. 1). After injecting 4 cc. of a 20 per cent aqueous radiopaque solution into the sinus, radiographs were taken. These films proved to be excellent myelograms in which the dye reached the thoracic vertebrae (fig. 2). Together with the clinical signs, they indicated that paresis must be due to a myelitis of the posterior portion of the spinal cord, no doubt associated with a meningitis.

Samples of blood and cerebrospinal fluid were collected for cellular examination and culture. The blood components were within normal limits and sterile. The cerebrospinal fluid was slightly cloudy and blood-tinged when collected, and had a leukocyte count of 500 cells per cubic millimeter, most of which were polymorphonuclear neutrophils. Cultures of this fluid produced colonies of *alpha* and *beta* hemolytic staphylococci that were sensitive to tetracycline.

The dog was put on a course of tetracycline. Its temperature was 104.0 F. on the second day, but by the next day had receded to 101.8 F., remaining normal for the duration of hospitalization. The dog's condition deteriorated and by the third day there was complete motor paralysis of the tail, little response to sensory stimuli, and the gait had become more unsteady.

On the third day, the sinus tract was excised. An elliptical skin incision was made around the opening of the sinus, and the

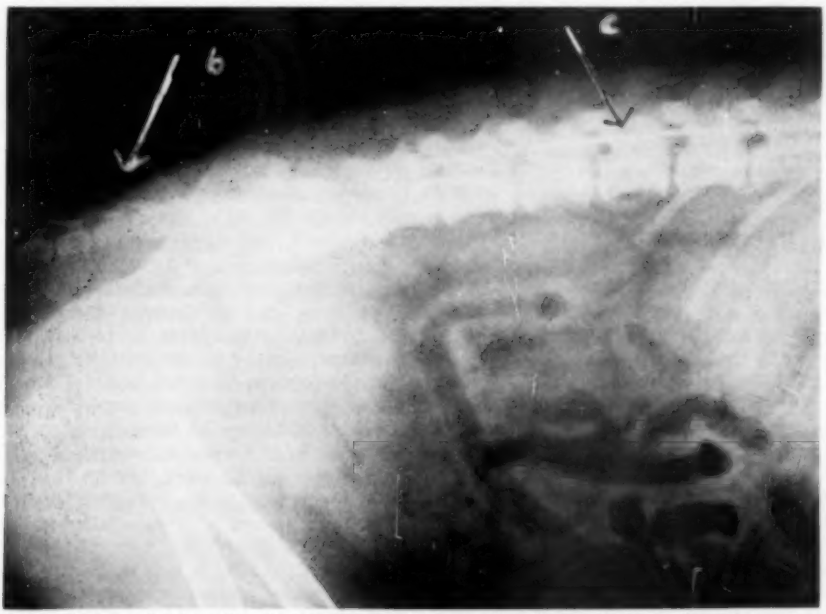

Fig. 2—Radiograph taken after the injection of radiopaque dye which outlines the dermoid sinus in a dog (b), and produces a retrograde myelogram (c).

tract dissected out by removing the laminae of the second and third sacral vertebrae, then ligating the tract as far as possible anteriorly. It was found that the sinus did not stop at the sacrococcygeal interspace, but extended to the first sacral intervertebral space. Histological findings of the sinus structure conformed to the description already given.

Postoperative antibiotic therapy consisted of penicillin-streptomycin for 13 days, even though the temperature remained within normal limits. On the sixth postoperative day, hyperesthesia of the sacral region was absent, and there was sensation and evidence of motor function in the tail. On the eighth day, the leukocyte count of spinal fluid had dropped to 360 cells per cubic millimeter. When discharged on the thirteenth day, the dog's gait was considered normal, and he was able to wag his tail.

SUMMARY

The hereditary occurrence of dermoid cysts in the cervical and coccygeal areas of Rhodesian Ridgeback dogs is reported.

A Canadian-bred Rhodesian Ridgeback that developed complications of a dermoid sinus was successfully treated by radical excision and appropriate antibiotic therapy.

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Polygamous Chinchilla Breeding.—To prevent females from injuring or killing the male chinchilla before they become acquainted, a rancher has arranged a series of cages for single females, each with a door which can be opened into a common runway for the male. The females wear lightweight plastic yokes large enough to prevent them from going through the door, when opened. If she still shows fight, the male can escape. After the 111- to 120-day gestation period, the litter arrives, the female is immediately rebred, and her door is closed. Thus each female raises 2 litters before the middle of September when she is either pelted or allowed to rest until December 1. In this manner, 1 male has served as many as 22 females.—*Nat. Chinchilla Breeder (Aug., 1957): 12.*

Oxytocin Speeds Farrowing.—Oxytocin was injected into 36 sows, at the University of Missouri, that were about to farrow (milk in their teats). Thirty of them delivered their first pig in an average of 3.5 hours and completed farrowing in another 2.2 hours. Control sows averaged half again as long. To be effective, the injection must be no sooner than 24 hours before normal time for parturition.—*Farm J. (Oct., 1957): 40.*

Ten Years of Artificial Insemination of Cows.—The artificial insemination program, started in 1938, is now serving at least 25 to 27 per cent of the milk cow population in this country. It has aided in the control of diseases commonly spread by natural service, such as *Vibrio fetus*, trichomoniasis, vaginitis, brucellosis, and leptospirosis, and has increased milk production per cow, as compared with naturally sired stablemates, by 225 lb. of milk and 8 to 9 lb. of butterfat. Also "shy breeding" cows are detected earlier and treated more promptly by veterinarians.

Of the 76 approved artificial breeding organizations in the United States, about 60 are cooperatives owned and operated by farmers. Approval by the Purebred Dairy Cattle Association means that the bulls must be identified; accurate records must be kept; and all semen must be accurately labeled and accounted for.—*West. Dai. J. (Sept., 1957): 29*

Parathyroidectomy of Cows and Parturient Paresis.—Seven nonlactating pregnant cows and 2 nonpregnant lactating cows, with no previous histories of parturient paresis, were partially or completely parathyroidectomized. A marked decrease in serum calcium and a smaller decrease in inorganic phosphorus occurred, but none of the cows showed signs of parturient paresis although the calcium sometimes decreased to the range at which this condition occurs. Thyroparathroidectomy in calves resulted in an abrupt lowering of the serum calcium, a less marked lowering of inorganic plasma phosphorus, tetany, and death.—*J. Dai. Sci. (Aug., 1957): 893, 897.*

Muscular Dystrophy in Calves and Lambs. I. The Relation of Environmental Conditions to Incidence

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MUSCULAR DYSTROPHY (white muscle disease) is responsible for unusual, though sporadic, losses in calves and lambs in limited small areas scattered throughout Idaho. This disease has been known to appear in many sections of the Rocky Mountain states. In Oregon, losses were reported in lambs in the early 1920's and in calves in 1939.[18]

REVIEW OF THE LITERATURE

Deficiency of vitamin E in the diet has been believed, for many years, to be the basic cause of muscular dystrophy in cattle and in sheep. Various workers[16,23-28] clearly demonstrated that the cause of stiff lamb disease (muscular dystrophy) was nutritional and that the feeding of a vitamin E source would prevent the condition. Recent experimental work has indicated that the mode of action of *alpha*-tocopherol in the prevention of muscular dystrophy is not clearly understood. Muscular dystrophy occurred in lambs from ewes which were fed 150 mg. of *alpha*-tocopherol per head per day for 30 days prior to lambing.[18]

The quantity of vitamin E in the ration has not been shown to be correlated with the signs of muscular dystrophy in either the calf or the lamb. It was postulated[2] that the abnormality arises from the synergetic action of tocopherol with some unknown environmental factor. The word "environmental" was used to imply a difference in nutrition and external stress such as weather and altitude.

The pathological conditions observed in vitamin E deficiencies of various animals have been believed to result from the function of lipid peroxides on sulfhydryl-sensitive enzyme systems. The production of lipid peroxides in the animal body are promoted by vitamin E antagonists such as unsaturated fats, o-cresyl esters,[3-5,9,14] carbon tetrachloride, pyridine sulfathiazole, sulfaguanidine, and sodium sulfite. These act as pro-oxidants.[14]

Investigators have demonstrated the following: Feeding tocopherol to the dam before pregnancy and to the calf following birth reduced the incidence of white muscle disease;[1] intramuscular injection of DL-*alpha*-tocopherol acetate caused no increase in the tocopherol concentration of the serum and resulted in only slight protection of the animal;[6] oral administration during terminal stages of pregnancy altered vitamin levels in the blood;[15] placental transfer of the vitamin was more difficult than the mammary gland transfer;[17] prepartum supplementation of cows did not markedly affect the serum tocopherol levels in the newborn;[19] muscular dystrophy occurred in calves on vitamin E-deficient diets.[20,22]

GEOGRAPHICAL DISTRIBUTION

Muscular dystrophy was recognized in calves in Idaho, in 1949, where the elevation was about 9,000 ft.; in both calves and lambs in the northwestern part of the state with an elevation of approximately 2,500 ft.; and in several other regions at 5,000 ft. where long winter seasons with considerable snow are prevalent.

Calves were reported by stockmen to have a condition referred to as "slipped shoulder" in 1940. One cattle owner lost 15 of 16 calves and 8 of the 15 showed "white muscle disease" at necropsy. Another owner lost 4 of 20 calves, which had lesions of "white muscle disease," during the spring of 1953; 9 of 27 calves in the spring of 1954; and 2 of 21 calves in 1955. No calves were lost during the 1956 season when the cows had been fed ½ to ¾ lb. of oats per day in addition to the roughage.

Another producer, who lost 15 of 16 calves with this disease in the spring of 1955, sold the dams and purchased a new group of cows which were fed hay grown in an entirely different area. In 1956, only 1 of 27 calves was lost and the cause of death was not muscular dystrophy.

Many deaths from muscular dystrophy have been reported in orphan calves that

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had received milk from cows whose calves had already died of muscular dystrophy.

All of the cases observed have been in beef calves. This would indicate that the ration of the dairy cow during pregnancy and lactation was more adequate than that of the beef cows. The dairy herds are fed a concentrate mixture while the beef herds receive only roughage. The signs of vitamin E deficiency in calves usually appear between the ages of 2 to 6 weeks but occasionally at 8 to 10 weeks of age.

Sudden heart failure appears to be the most common cause of death. The first warning sign, generally, is difficult breathing. Close observation will reveal abdominal breathing with an absence of movement in the chest wall during respiration. Breathing is generally accompanied by an expiratory grunt.

All forms of paralysis, from slight lameness to complete inability to stand, are commonly seen. Usually, the first sign observed is the reluctance of the calf to stand quietly in any one position. The characteristic posture for the calf is standing with its forelegs bent at the knees. Occasionally, the calf will attempt to stand on its toes. If the muscles of the shoulder blades are affected, there is a tendency toward the development of a hollow space between each scapula and the body. Many cattlemen have referred to this condition as "dropped shoulder."

Many cases have been reported in which the tongue becomes paralyzed and the calf is unable to grasp food. If the throat is affected, the calf may have difficulty in swallowing and may choke while drinking. In general, the signs of the disease in calves in Idaho are similar to those reported elsewhere.[7,10-13]

NECROPSY FINDINGS

The observed lesions involve both heart and skeletal muscle. The muscle fibers show degeneration or necrosis, and the condition can be seen readily as white areas in a normally reddish muscle. If the skeletal muscles are affected, identical muscles on either side of the body show degeneration. If the heart muscle is affected, large white areas or streaks are visible to the naked eye. Associated with muscle degeneration is an accumulation of watery fluids in the lungs and in the pleural and pericardial cavities.

FEED STUDIES

It is extremely doubtful that a deficiency of vitamin E (tocopherol) exists in the areas of Idaho where the disease is unusually prevalent, since there is considerable evidence that feeds available for cattle and sheep are relatively high in tocopherol. During the fall of 1950, samples of feeds from ranches where the incidence of the disease was unusually high were analyzed for tocopherol content* (table 1). The total tocopherol, the *gamma*-tocopherol, and the *delta*-tocopherol contents were determined. The *alpha*-tocopherol content of the feeds is computed by subtracting the total of the *gamma*- plus the *delta*-tocopherols from the total tocopherol values.

TABLE 1—Tocopherol Content of Samples Taken in Valley County, Idaho, 1950

Sample No.	Feed	Total tocopherol content μg./Gm.*	Gamma-plus delta-tocopherol content μg./Gm.	Alpha-tocopherol content μg./Gm.
1	Chopped oats	31.2	3.1	28.1
2	Alfalfa-timothy-red top-brome grass-hay mixture	149.2	15.2	134.0
3	Alfalfa hay (frosted 1949 crop)	132.6	10.4	122.2
4	Alfalfa-brome-grass-timothy mixture	79.8	10.4	69.4
5	Timothy-alsike-clover-red-top hay mixture	96.1	7.3	88.8
6	Timothy-blue-grass-red-top-white-dutch-clover hay mixture	154.0	17.4	136.6

*Micrograms of tocopherol per gram of feed.

A relatively wide variation was shown in the total tocopherol content of the samples analyzed. The lowest content was observed

*Credit is due Dr. Phillip Eldredge, veterinarian, Emmett, Idaho, for a study of the conditions and the pathological changes in the calves involved, and for collecting the feed samples for analysis. Also to Dr. John W. Bratzler, Department of Animal Nutrition, Pennsylvania State University, for the determination of the tocopherol content of the samples.

in the ground oats grown on irrigated soil (sample 1). However, these values are not considered sufficiently low to produce vitamin E deficiency in cattle. The highest quantity of tocopherol was in the hay mixture of timothy, blue grass, red top, and white dutch clover (sample 6) which had been grown on irrigated land, harvested, and stored for one year before being analyzed. Since the third highest content of total tocopherol (sample 3) was in frosted alfalfa hay, which had been stored for one year, it is apparent that the storage and freezing of hay does not destroy the tocopherol content. The hay in sample 3 had been grown on nonirrigated soil and growth was stopped when it was frosted during the middle of June.

The lowest total tocopherol value, 79.8 μg. per gram of hay, was in alfalfa-bromegrass-timothy mixture (sample 4) grown at an altitude of 5,000 ft. on a nonirrigated sandy loam soil which was low in phosphorus. The annual rainfall was about 20 inches.

The second highest total tocopherol value, 149.2 μg. per gram of hay (sample 2), was in a mixture grown on both irrigated and nonirrigated land, while the second lowest total tocopherol value, 96.1 μg. per gram, was in a mixture (sample 5) grown on nonirrigated soil.

There was a wide variation in the tocopherol values of feeds consumed by the pregnant cows. The quantities reported in all samples have been considered as sufficient to meet the theoretical needs of pregnant and lactating cows and ewes. Since the incidence of muscular dystrophy in calves produced by these cows was high, the tocopherol values are thought to be of doubtful value in determining the cause of the disease.

Brown[8] made a similar study on the chemical determinations of the total tocopherol content of feeds. He questioned the value of chemical determinations since there appeared to be no relationship between the incidence of signs of vitamin E deficiency and total tocopherol content of feeds.

SUMMARY

Muscular dystrophy (white muscle disease) appeared in calves and lambs from dams fed and wintered within specific areas of the Rocky Mountains.

The disease appeared in calves and lambs whose mothers had been fed roughages without concentrates.

The incidence of the disease was high in calves whose dams had received 79.8 to 154 μg. of tocopherol per 100 Gm. of hay. This would indicate that factors other than a tocopherol deficiency are responsible for the muscular dystrophy in calves.

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Antibiotic Combinations.—Since the introduction of erythromycin, other antibiotics with similar antibacterial spectrums have been encountered, none of them equal to erythromycin. Three have been commercially produced. The first, carbomycin (Magnamycin), has been almost completely abandoned and the other two—oleandomycin, in this country, and spiramycin, in France, have been introduced in combinations. There is marked or complete cross resistance to each in strains of organisms made highly resistant to any of them. No combination of these antibiotics with tetracycline was superior to either component used alone.

Studies of the antibacterial action of blood, after ingestion of the various antibiotics, indicate that the new ones are sufficiently inferior to erythromycin so that their use in general should be discouraged. Furthermore, the use of these antibiotic combinations "is not justified . . ., represents bad practice, and is not in the best interest of the patient."—*New England J. Med. (Sept. 12, 19, 1957):* 481, 536.

Clinical Assessment of Penicillin V.—Phenoxymethyl-penicillin (penicillin V) which, unlike benzylpenicillin (penicillin G), is stable in gastric juice, was used orally in capsule or tablet form (120 mg., 4 times daily in adults) in 110 cases in a British hospital. Results were no less satisfactory than when penicillin was given intramuscularly. Two cases of cutaneous anthrax in bone-meal workers responded satisfactorily. It was equally effective in staphylococcic infection.—*Brit. Med. J. (July 27, 1957): 191.*

Hydrocortisone and Cleft Palates.—When hydrocortisone was given to mice on the twelfth and thirteenth days of gestation, about 50 per cent of the offspring had cleft palates when the dose was 5 mg.; 64 to 100 per cent when the dose was 10 mg. When given before the tenth or after the sixteenth day of gestation, no defects followed. Two cases of cleft palate in children were reported when the mothers were given cortisone therapy during the first two months of pregnancy.—*Nutr. Rev. (Oct., 1957): 318.*

More on Anthrax in Dogs.—In 1955, 3 adult Foxhounds in one kennel, in England, died with confirmed anthrax four days after being fed raw beef obtained at a rendering plant. The dogs showed dullness and depression for three to 12 hours before death, but no head swellings.—*Vet. Rec. (Sept. 14, 1957): 893.*

Anthrax in Sweden.—During the 15 months prior to April, 1957, anthrax appeared on 68 premises, with fatalities in 72 swine and 19 cattle, the majority probably caused by contaminated meat meal. Of 506 meat meal samples examined, *Bacillus anthracis* was found in 45 (8.7%).—*Nord. Vet.-med. (Sept., 1957): 641.*

The Use of an Adsorbed Swine Erysipelas Bacterin—A Controlled Field Test

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THE CLINICAL SIGNS, morbidity, and mortality of swine erysipelas are extremely variable, and recurrence of the disease on any premises is unpredictable. Mortality is often low, but considerable economic loss frequently results from stunting and unthriftiness, and from arthritis in the chronic form of the disease.

The main problem in experimental work with swine erysipelas has been the inability to reproduce consistently the same disease syndrome as seen in the field. While some workers[2-6,9] have been able to reproduce the characteristic febrile, systemic, and skin reactions with the scarification method time after time, others[7] have not. When either 10 cc. or 20 cc. of a 24-hour *Erysipelothrix rhusiopathiae* culture has been inoculated intravenously,[7] death frequently resulted. Since mortality in natural field outbreaks is often low, the infection caused by intravenous challenge may be more severe than naturally occurring infections. The same difficulty is encountered with the scarification method,[3] although the infection it causes is not as severe as that following experimental intravenous infection. However, the scarification method has been used more frequently because signs of erysipelas can be reproduced without causing as high a mortality.

Under experimental conditions, when 6 pigs vaccinated with one 5-cc. dose of an adsorbed erysipelas bacterin (Duovax*) were challenged intravenously six weeks postvaccination, some protection existed, since all survived challenge and showed only a mild febrile reaction.[8] However, all pigs developed chronic polyarthritis, and no protection was evident at further challenge 12 weeks postvaccination. In contrast,[5] when the scarification challenge was used three months after vaccination, protection was shown in 5 per cent of the pigs vaccinated with anti-swine erysipelas serum and *Ery. rhusiopathiae* culture, and in 55 per cent of the pigs given one dose of bacterin. Seventy two per cent of the pigs were protected for a four-month period after having been given a second 5-cc. dose of bacterin one month after the first.

On farms in five midwestern states where erysipelas had previously been a serious problem and where 9,210 swine were routinely vaccinated with erysipelas bacterin, results seemed favorable.[1] However, no pigs were left as unvaccinated controls; therefore, the bacterin's efficacy in protecting the pigs to market age was only presumed.

Since experimental results have differed so widely, the present controlled study was undertaken in the field, where erysipelas was enzootic, to evaluate the use of the erysipelas bacterin under natural conditions.

PROCEDURE

This field trial included a total of 4,292 pigs from 50 herds on farms, mostly in Missouri, where erysipelas had been a problem for many years. All pigs were farrowed from sows that had been vaccinated against erysipelas, or had been raised on erysipelas-infected premises, or both.

Each pig was individually identified by ear notching and tagging. Before vaccination, the pigs were examined clinically for disease and then were randomly allotted to the three experimental groups in each herd. At approximately 6 to 10 weeks of age, all of the pigs were vaccinated against hog cholera. Virulent hog cholera virus and anti-hog cholera serum or modified live virus hog cholera vaccine alone, or with antiserum, were used according to the owner's preference. The use of anti-hog cholera serum in some herds might have been responsible for the low incidence of erysipelas in this field trial. However, comparisons were made within herds, so these comparisons are valid.

Approximately one third of the pigs in each herd were retained in the unvaccinated control group. The remaining two thirds were allotted to two groups; those in one group were vaccinated with a single dose of erysipelas bacterin, and those in the other with two doses, the second dose given three to six weeks after the first. The bacterin was in-

From the American Cyanamid Co., Pearl River, N. Y.

*Duovax, the tradename for erysipelas bacterin, is produced by the American Cyanamid Co., Pearl River, N. Y.

The author thanks the following veterinarians for their cooperation: Harold E. Bain, Cameron, Mo.; M. D. Conrad, Plattsburg, Mo.; Donald R. Fields, Monona, Iowa; V. D. Goodknight and Robert E. Gouge, Sedalia, Mo.; and Carl Hulen, Columbia, Mo.; and Mr. Jack Haynes for statistical analysis.

TABLE 1—Occurrence of Erysipelas in Pigs by Age Groups

Age at vaccination (wk.) First	Second	Total No. Herds	Pigs	No. herds developing erysipelas	No. pigs in these infected herds	Erysipelas mortality	Erysipelas morbidity Acute[3]	Chronic[1]
Group 1		2		0				
Unvac. controls			37		0	0	0	0
1-1½	-		39		0	0	0	0
1-1½	7-9		39		0	0	0	0
Group 2		2		2				
Unvac. controls			40		40	7	12	8
1½-2	-		41		41	2	6	7
1½-2	6-7		41		41	0	2	2
Group 3		7		1				
Unvac. controls			159		37	1	14	12
2	-		159		38	0	15	3
2	6 or 10		159		38	0	3	3
Group 4		3		0				
Unvac. controls			57		0	0	0	0
2-6	-		73		0	0	0	0
2-6	6-10		75		0	0	0	0
Group 5		12		1				
Unvac. controls			356		58	0	0	3
3-8	-		352		59	0	0	5
3-8	7-12		351		59	0	0	1
Group 6		12		4				
Unvac. controls			422		171	8	26	10
5-6	-		438		171	0	0	3
5-6	8-12[2]		340		81	0	0	0
Group 7		12		2				
Unvac. controls			375		18	0	0	3
Over 6	-		392		17	0	0	0
Over 6	Approx. 4 weeks later		347		17	0	0	0
Totals		50	4,292	10	886	18	78	60

[1]Includes those pigs that developed chronic erysipelas following the acute disease. [2]In this group, 11 herds received two doses, and one herd one dose. [3]Includes 31 control pigs given anti-swine erysipelas serum.

TABLE 2—Summary of Erysipelas Morbidity and Mortality

No. of doses of bacterin		0 (Control)	1	2
Total No. pigs		324	326	236
Acute erysipelas				
Total incidence	(No. pigs)[1]	68.0	23.0	5.0
	(%)	20.9	7.0	2.1
Mortality	(No. pigs)	16.0	2.0	0
	(%)	4.9	0.6	0
Survival	(No. pigs)	52.0[2]	21.0	5.0
Chronic erysipelas				
Total incidence	(No. pigs)	36.0	18.0	6.0
	(%)	11.0	5.5	2.5
Acute cases that became chronic	(No. pigs)	23.0	9.0	5.0
Chronic cases that were never acute	(No. pigs)	13.0	9.0	1.0
	(%)	4.0	2.8	0.4
Total incidence of erysipelas	(No. pigs)[3]	81.0	31.0	6.0
	(%)	25.0	9.5	2.5

[1]Incidence of acute erysipelas between (a) control and vaccinated animals differs significantly at $P<0.0001$; (b) 1 and 2 doses differs significantly at $P=0.014$.

[2]Thirty one of these pigs were given anti-swine erysipelas serum, and only 3 developed chronic erysipelas.

[3]Total incidence between (a) control and vaccinated animals differs significantly at $P<0.0001$; (b) 1 and 2 doses differs significantly at $P=0.002$.

jected subcutaneously on the inside of the flank. The ages at the time of first vaccination ranged from 1½ to 12 weeks. Pigs that were 2 weeks old or younger were given 3 ml. of the bacterin; pigs 2 to 6 weeks old were given either 3 or 5 ml.; pigs older than 6 weeks were given 5 ml. (table 1). They were marketed at approximately 6 to 8 months of age.

Necropsies were performed on all animals that died, and routine bacteriological examinations were made of tissue specimens of heart, spleen, liver, lungs, kidneys, and joints. Blood samples were taken from all sick animals for laboratory studies to confirm clinical diagnosis. Chronic erysipelas was diagnosed by observations of clinical signs of arthritis.

RESULTS AND DISCUSSION

In this field trial, erysipelas occurred in ten of the 50 herds, containing 886 of the 4,292 pigs (table 1). Eighteen (2%) of the animals in the infected herds died. A total of 16 deaths (4.9%) occurred in the 324 control pigs (table 2). No mortalities occurred in the 236 pigs given two doses of bacterin, and only two deaths occurred in the 326 pigs that were given one dose of bacterin. These losses were in the group vaccinated at 1½ to 2 weeks of age.

Acute erysipelas was observed at various times throughout the field trial, primarily

in pigs vaccinated at an earlier age. The greatest incidence was confined to the control pigs and to the pigs given one dose of bacterin. Acute erysipelas developed in 20.9 per cent of the control pigs. This was significantly greater than the 7.0 per cent of the pigs vaccinated only once; and the latter was significantly greater than the 2.1 per cent of disease in the pigs given a second dose of bacterin.

Chronic erysipelas was observed in 11 per cent of the control pigs, in 5.5 per cent of the pigs given one dose of bacterin, and in 2.5 per cent of those given a second dose of bacterin. The incidence of chronic erysipelas in the control group was undoubtedly reduced by the administration of a therapeutic dose of anti-swine erysipelas serum to 31 of the 52 acutely affected pigs, since only 3 of these 31 pigs developed chronic erysipelas as compared with 20 of the 21 pigs not given serum. Of the total number of pigs in which acute erysipelas was not observed, chronic erysipelas occurred in 4.0 per cent of the control pigs, in 2.8 per cent of the single-dose group, and in 0.4 per cent of the two-dose group.

The total incidence of erysipelas (table 2) which occurred under conditions existing on the ten farms involved, was 25 per cent in the control pigs, 9.5 per cent in the pigs given one dose of erysipelas bacterin, and 2.5 per cent in those given two doses.

SUMMARY

A total of 4,292 pigs in 50 herds on farms in the southwestern portion of the corn belt, where erysipelas had been a problem, was included in a field trial to evaluate an adsorbed swine erysipelas bacterin. Approximately one third of the pigs in each herd served as unvaccinated controls. One half of the remaining two thirds were vaccinated with a single dose of adsorbed erysipelas bacterin and the other half were given a second dose three to six weeks following the first. Erysipelas occurred in ten of the 50 herds, containing 886 pigs, from three weeks postvaccination until marketed at 6 to 8 months of age. Erysipelas, either acute or chronic, occurred in 25 per cent of the unvaccinated control pigs, in 9.5 per cent of the pigs given a single dose of the bacterin, and in 2.5 per cent of the pigs given two doses. Erysipelas, either acute or chronic, among the vaccinated pigs, was largely confined to those pigs given one dose at an early age; acute erysipelas occurred only in pigs given the first dose (usually the only dose) of the bacterin at 2 weeks of age or younger. Significant protection was conferred by one dose of bacterin, but a definitely superior protection resulted from a second dose.

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Botulism in Mink.—In June, 1957, 5,981 (46.25%) of 12,932 mink on 20 ranches, in Washington, died of botulism. All were fed the same cooperatively, freshly prepared feed, and most of the deaths occurred on the following day. One rancher lost 432 of 722 unvaccinated mink, but none of 14 vaccinated females or their kits. Another lost 3 animals, the only ones that were not vaccinated.—*J. R. Gorham in Fur News (Oct., 1957): 12, 46*

Histological tissue changes (central nervous system, lymph nodes, spleen, and liver) were milder in pigs inoculated with lapinized hog cholera virus than in those with natural infection.—*Vet. Bull. (July, 1957): Item 2077.*

Unusual Tuberculous Lesion in a Wallaby

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THE MARSUPIALIA and the Herodiones were the only important orders in which no cases of tuberculosis had been found, in 1923, in the collection of the Philadelphia Zoological Garden.[2] In 1925, a tuberculous kangaroo (rufous rat kangaroo, *Aepyprymnus rufescens*) was reported from the London Zoological Garden.[6] Discrete miliary abscesses were present in the liver, and acid-fast bacteria were found in large numbers in the greenish pus from an enlarged mesenteric lymph node. In animal tests, severe generalized tuberculosis developed only in the rabbit and hen, indicating that the strain was the avian type of *Mycobacterium tuberculosis*. However, this organism was cultured from a lymph node of a guinea pig after three blind passages in that animal.

In 1928, in cultures from a series of tuberculous captive wild mammals,[6] another small rat kangaroo with extensive pulmonary lesions was found to be infected with the bovine type of tubercle bacillus. A Kangaroo Island kangaroo (*Macropus fuliginosus*), which seemed in good condition, died in the London Zoo; it had an enlarged axillary node full of creamy pus and a shoulder joint which was distended with pus, with erosion of the articular ends of the bones.[6,9] The lungs, the lymph nodes generally, and the sternoclavicular joints were also involved. Tests in animals showed that the organism was of the avian type.

The first case of tuberculosis observed among 119 Macropodidae in the Philadelphia Zoo was reported in a black wallaby (*Macropus ualabatus*) in 1935.[3] The following year, another case was reported in a kangaroo (*Macropus robustus cervinus*).[4] The type of tubercle bacillus was not ascertained.

The following types of *Myco. tuberculosis* were reportedly recovered in a bacteriological study of a large number of captive wild animals: (*a*) Ungulata (15)—bovine type 11, human type 4 (the infection was usually respiratory); (*b*) monkeys (30)—human type 22, bovine type 8 (monkeys seem not to be susceptible to avian tuberculosis); (*c*) carnivora (2)—1 bovine, 1 human type; (*d*) birds (17)—all avian type.

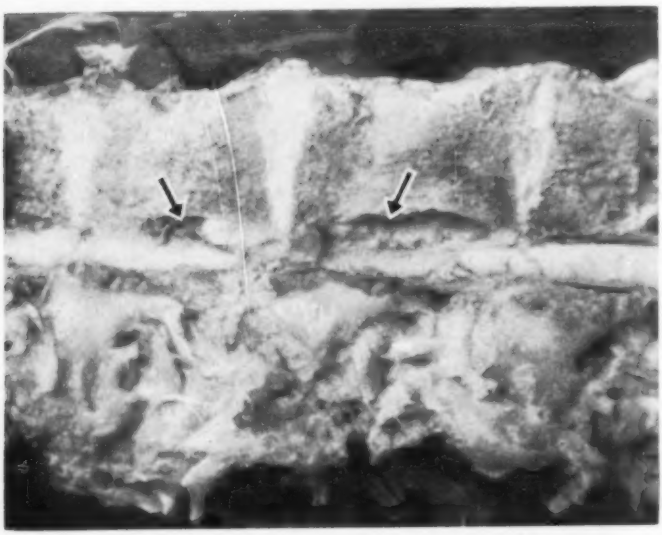

Fig. 1—Sagittal section of the vertebral column in a wallaby showing compression of spinal cord at the site of the abscess (arrows) which was located at the level of the third and fourth lumbar vertebrae.

From the Animal Hospital, Chicago Zoological Park, Brookfield, Ill.

The frequency of the human type of tuberculosis in captive wild animals and birds was mentioned in 1930.[7] It was reported to be prevalent in pet parrots.[1]

Other isolations reported[7] were: bovine type from the tapir, reindeer, and white-tailed gnu; and human type from the two-spotted palm civet, orangutan, and chimpanzee. All reported recoveries of the tubercle bacillus from various animals, domesticated and wild, have been tabulated.[10]

In addition to the rarity of tuberculosis in the kangaroo, the following case is unusual in that the single lesion was a tuberculous abscess in the spinal canal (fig. 1). The animal, a young, well nourished, male wallaby (*Macropus bennetti*) was unable to stand when released from the shipping crate when recently acquired, but it showed no injuries. It did not improve and, since muscle dystrophy in tree kangaroos, due to deficiency of vitamin E, had been reported,[5] it was given vitamin E intramuscularly and multiple B vitamins parenterally. At necropsy, after it was unexpectedly found dead, an abscess was found in the spinal canal, involving the first and second lumbar vertebrae. Except for passive congestion of the lungs, no other noteworthy changes were observed.

The pus yielded no growth on blood agar plates, but an acid-fast stain revealed numerous tubercle bacilli. The pus was collected, after 48 hours on the two blood agar plates, by washing it into a centrifuge tube with 23 per cent tertiary sodium phosphate. After standing overnight at 4 C., the sediment after centrifugation was washed twice with sterile saline solution, then planted on Lowenstein and Petragnani slants.* After 30 days, some roughness of the surface of the Petragnani slant appeared, and staining showed that acid-fast bacteria had developed. No growth appeared on the Lowenstein slant, but subcultures from the initial tube grew out on both mediums within three weeks.

The smoothness of the growth suggested that the organism was of the avian type of *Myco. tuberculosis*, and this conclusion was confirmed† when the culture proved pathogenic for rabbits and chickens but induced no infection in guinea pigs.

SUMMARY

A tuberculous abscess in the spinal cord of a wallaby was found to be due to the avian type of *Mycobacterium tuberculosis*. Only a few reports of tuberculosis in this type of animal were found in the literature. In the 3 cases in which the organism was cultured, 1 was of bovine type, the other 2 of avian type.

*Difco Laboratories, Detroit, Mich.

†The culture was identified by the Communicable Diseases Center, Chamblee, Ga.

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Congenitalness of Toxoplasmosis in Man.—Three cases of infection with *Toxoplasma gondii* are described, one of them in a pregnant woman. She delivered a normal baby which had a strong dye test titer at first, but it gradually receded. A report is cited of 45 mothers who had given birth to congenitally affected children, but subsequently had 67 children with no evidence of toxoplasmosis. The inference is that passive immunity can be transferred from the mother to the child.—*Brit. Med. J. (July 20, 1957): 141.*

Leptospira and Periodic Ophthalmia.—Leptospira were recovered, in Argentina, from laboratory animals injected with samples of aqueous and vitreous humor from the eyes as well as from the urine, kidney, spleen, and liver of horses with periodic ophthalmia. The high agglutinating titer and icteric color of the humors were believed to indicate that the eye is a reservoir of infection.—*Vet. Bull. (Sept., 1957): Item 2626.*

Nutrition

Modern Day Nutrition— A Roadblock or an Opportunity?

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Livestock farming today is big business —a changing business. Our nation's agricultural assets, according to the U.S. Department of Agriculture, totaled about $176 billion. The average dollar investment per farm totals $36,000, with the investment on many farms exceeding $100,000.

The trend is to a highly mechanized agriculture with fewer farm workers. In 1910, 35 per cent of our population was on the farm. In 1956, only 13 per cent of the population was farming. By 1975, if this trend continues, only 7.5 per cent of the population will be engaged in feeding the rest of the 205 million people, an anticipated population increase of 25 per cent.

To meet this need, we will require an annual slaughter of 10 million more cattle and calves, 20 million more hogs. We will need another 6 million milk cows, or the annual rate of production per cow will have to go up to 1,400 lb. Another 250 million broilers will be necessary. We will need 90 million more laying hens, or each bird will have to contribute an additional 50 eggs each year.

Only one fact remains static. Livestock and poultry farming is still strictly carried on for only one reason—to convert feedstuffs to food.

The veterinarian, whether he be engaged in practice, research, or any other vital phase of veterinary medicine, has always been an important partner to the farmer in the fight against costly disease. Today, in this competitive, accelerated agriculture, the battle lines against disease can no longer depend solely on defensive action. They must also show an offense; there is a marked swing toward preventing disease, rather than only curing it. This involves better breeding, improved sanitation and management, and modern nutrition methods.

Today, the veterinarian finds that knowledge of nutrition can be of tremendous value in the prevention, diagnosis, and treatment of disease. In Wisconsin, it has been estimated that over 50 per cent of veterinary calls to dairy herds are for noninfectious diseases.

In this new agriculture, there have been almost unbelievable improvements in efficiency of production. Evidence of this is the fact that, 25 years ago, each farm worker produced enough food for himself and nine other persons; now he produces enough for himself and 18 others. Today, the modern beef steer grows as much in one year as the old Texas Longhorn grew in three years. The modern hog makes as much growth in five to six months as the old razorback made in two years. Modern hens, laying 250 eggs a year, produce four times as many eggs as the average farm hen did 50 years ago.

Between 50 to 80 per cent of the costs of producing meat, milk, and eggs are feed costs. The feed industry has taken basic research in animal nutrition from experiment stations, has applied it, and has made possible this tremendous production increase. The amount of feed needed to raise a broiler has been cut almost in half. It takes about 3 lb. less feed to produce a dozen eggs than it did in 1930. Growing and fattening a hog—from weaning to marketing—requires 200 lb. less feed than it did 25 years ago. Feed is also being used far more efficiently in milk and beef production. This increased feed efficiency has cut the amount of feed required for our annual production of livestock and poultry by hundreds of thousands of tons! In so doing, it has also cut the livestock farmer's feed costs by millions of dollars.

The need for our work is evidenced by the feed industry's dynamic growth. Today, the feed manufacturing industry is ranked ninth among all manufacturing industries in this country. It is the largest manufacturing industry serving agriculture.

In order to keep every animal growing and producing to the limit of its ability, the animal must be kept strong, healthy, and disease-resistant. Increased production puts increased demands on livestock feeding rations. Unbalanced and deficient rations, still used on many farms today, result in slow gains, low production, and wasted feed.

Frankly, both the feed industry people and the veterinarians have made mistakes in the past, but let's look ahead to some of the roadblocks that we can avoid in our effort to help the American farmer.

The veterinarian should increase his

Dr. Spitzer, Burlington, Wis., is a past chairman, Nutrition Council, American Feed Manufacturers Association.

store of knowledge concerning modern day feeds and feeding. He would profit by cooperating and working closely with qualified nutritionists and feed men. Up-to-date nutritional information can be of value in the diagnosis, treatment, and prevention of animal diseases.

On the over-all picture, more diagnostic laboratories, more research, more disease prevention, more understanding, better communication all could be helpful.

The feed industry and the veterinary profession are making great strides toward the enactment of a sound working partnership. The AVMA has a nutrition committee; there is a representative of the veterinary profession on the nutrition council of the American Feed Manufacturers' Association; and a new group of veterinary nutritionists is extremely interested in nutrition. There is an ever increasing interchange of personnel from both our fields.

All this is serving to strengthen this partnership—a partnership with a common purpose—service to animal health, research, and livestock production.

Fuming Silos.—In recent years, many silos have produced enough nitrogen dioxide gas to kill people or animals. This seems to result from the ensilaging, during hot weather, of forage with a higher-than-normal nitrate content. The gas which results from oxidation products of the nitrates ranges from colorless to yellow, red, brownish, or even black. It has a sharp penetrating odor and is so heavy that it settles into low places. Upright silos are most dangerous, especially when they are part of a cluster of buildings. The greatest danger is while filling the last half of the silo and for ten days thereafter. The fumes may be colorless until agitated by a blower. They kill the forage on the down-slope from the silo. The silage may be too acid to feed until much later than usual.—*A. A. Case in the Sheep Breeder (Oct., 1957): 6.*

Mesquite Bean Malnutrition in Cattle.—In Texas, in 1955, perhaps 5,000 cattle developed "jaw and tongue trouble," apparently due to eating mesquite beans. Affected animals salivate profusely, chew continuously, often hold their heads sideways and, when they regurgitate, beans and other rumen content may be thrown over a wide area. In some animals, the tongue may protrude, and large sublingual or submaxillary edematous swellings may occur. The cattle are usually anemic and emaciated but have a fair appetite. Mesquite beans have been considered an excellent feed but, when eaten continuously for two months or longer, the sugar in the beans apparently changes the bacterial flora in the rumen so that cattle can not digest cellulose or synthesize the B vitamins. They can digest good concentrates but not hay. Affected animals will partially recover if grazed on lush green pasture. Treatment is of little benefit.—*J. W. Dollahite et al. in The Cattleman (Oct., 1957): 30.*

Grape Poisoning in Cattle.—Dairy cows which have consumed excessive amounts of grapes, particularly fresh grapes, in the grape-growing areas of Australia, develop a characteristic syndrome. They show anorexia, ruminal and intestinal stasis, tachycardia, tenesmus, and posterior paralysis and, when illness develops within 12 to 24 hours, it is nearly always fatal. Cows with subacute (72 hour) cases may gradually recover. Purgatives are of little value. The best therapy is an injection of atropine sulfate (0.5 gr.) ten minutes before an injection of acrecoline hydrobromide (1.0 gr.) or lentin (5.0 mg.). Rumenotomy is indicated with this therapy in severe cases.—*Austral. Vet. J. (Aug., 1957): 210.*

Equine Photodermatitis.—A mare developed photodermatitis with skin necrosis on the only white-haired area on her body—the right hind fetlock; she also showed conjunctival discharge, excitability, and a disturbed gait and equilibrium. She was believed to have "hypericine" poisoning from eating the green plant, *Hypericum maculatum.*—*R. Vind in Nord. Vet.-med. (April, 1957): 322.*

Gibberellic Acid and Plant Viruses.—Gibberellic acid, a growth stimulant which is effective against certain dwarfisms in plants, was shown to be effective against three leafhopper-borne virus diseases in plants. The reversal of stunting in sweet corn, infected with a corn-stunting virus, was visible in 48 hours, but recovery from wound tumor virus in crimson clover was not evident until day 5.—*Science (Oct. 4, 1957): 651.*

Editorial

Washington State V.M.A. Employs an Executive Secretary

The reorganization of the Washington State V.M.A. and its recent action in employing a full-time executive secretary are noteworthy in several ways.

First—this association has an active membership of only about 300 veterinarians, a rather small number compared with other associations having full-time secretaries: California (1,000); New York (800); Ohio (750); and Texas (500).

Second—all expenses of the association, including those of the executive secretary, will come from membership dues, although other sources are being explored.

Third—the reorganization was completed within three years of its inception. We recall the enthusiasm and sincerity of veterinarians who served on the local committees for the AVMA meeting in Seattle, in 1954, and would like to believe that the flame of reorganization was kindled then.

Those who join the ranks of the Washington V.M.A. in the future may be inclined to ask why the association was reorganized. The answers should not be hard to find. Association membership benefits, tangible and intangible, are generally recognized as good reasons for belonging.

Associations are founded on the desire of individuals to solve their common problems by working together. Even though veterinarians often have been characterized as "rugged individualists," and though they usually practice as individuals, they sense a responsibility to the profession that can best be discharged at the local level. They also recognize that it takes money to accomplish this. Therefore, the members of the Washington State V.M.A. raised their annual dues to $35.

This figure was arrived at by drafting a budget to cover the things they felt they should and could do—and dividing the total by the number of members. They were not concerned about the dues of other state associations. They knew that they were still charging themselves less than the bricklayers, plumbers, and truck drivers were paying as organization dues.

Message of the President

The address of the retiring president, Irwin Erickson, apparently reflects the attitude of veterinarians in his state as to the benefits of organization. Excerpts of the address follow:

We are pleased to pay tribute to all veterinarians—past and present—who have made this organization possible.

The respect that we have for each other's part in the profession, our moral sense of unity, our dedicated devotion to a common cause, the broad cause of animal health, our sense of service in the work that we do—these things will largely determine our future and our acceptance as a profession and as individuals. These things will also largely determine our worth to our nation and to ourselves in this restless and rapidly shrinking world, with the dark threats of transmissible animal diseases ever present.

We know there will be many problems with veterinary organizations in general and indeed with the Washington State Veterinary Medical Association. This is the price of progress. The knowledge of animal health, the physical equipment used by today's veterinarians, the increasing investment that today's veterinarian has in his profession have all increased greatly. These dramatic changes have been accepted but with them comes a renewed need for progress in veterinary organization so that it may catch up with the progress made in the other professional activities.

To treat animal disease; to reduce animal suffering; to plan and administer animal health programs; to teach and orient the young people who become our colleagues; to find new truths about animal diseases; and to protect the human population from the direct and indirect effects of animal diseases—these are all a part of the growing challenge. To solve these problems and many others, and to successfully cope with the competing pressures which face the profession, will require the mature wisdom of our pioneers, the support of all present-day veterinarians, and the helpful hands of our future colleagues.

We will do our best as a profession when we are truly united and working for a common cause. Thus, we will improve our communications within the profession, with allied healing professions, and with the entire public. Thus, we will elevate and unite the veterinary profession.

Incidence vs. Prevalence of a Disease

Incidence is defined as the number of new cases of a disease occurring within a specific period of time, whereas prevalence is defined as the number of cases of a disease present in the population group as of a specified interval of time, that is, the number of cases existing plus the new cases developing during the interval. In more formal terms, incidence measures the rate at which new cases are added while prevalence is a function of (depends on) the incidence rate and duration of a disease.—*Am. J. Pub. Health (July,1957):826.*

ABSTRACTS

Disease of Cattle and Swine Caused by Moldy Corn

Thirteen cultures of mold were isolated from corn gathered in fields where swine were dying. Two of the 13 cultures proved toxic when grown in a pure culture on autoclaved corn. Experimental poisoning was produced in swine and mice with toxic *Aspergillus flavus* Link and *Penicillium rubrum* Stoll. A goat and 4 horses were also poisoned with *P. rubrum* Stoll. The toxicity of these two molds was further confirmed with tests on mice and embryonating eggs, and antibacterial tests with culture filtrates on selected organisms. The gross histological lesions produced by the toxic molds are discussed.—[*J. E. Burnside, W. L. Sipple, J. Forgacs, W. T. Carll, M. B. Atwood, and E. R. Doll: A Disease of Swine and Cattle Caused by Eating Moldy Corn. II. Experimental Production with Pure Cultures of Molds. Am. J. Vet. Res., 18, (Oct., 1957): 817-824.*]

Immunization of Cattle Against Anthrax

Two subcutaneous injections of alum-precipitated antigen produced immunity to anthrax in cattle. The resistance was effective one month after immunization, had declined somewhat after three and a half months, and was barely detectable after seven months. One challenge dose of 1.5 x 10^4 spores, given by mouth, was fatal to 74 per cent of the control animals. In animals that died of infection, a characteristic lesion was consistently found in the small intestine and adjacent mesenteric lymph nodes. This lesion appeared to be the portal of entry for the generalized infection. A single injection of alum-precipitated antigen was less effective than two injections.

The effectiveness of a nonencapsulated living spore vaccine was also tested in a single experiment. All of 6 animals survived challenge three months after immunization, but all showed evidence of infection. Characteristics of the disease in normal and partially immune animals are discussed, and the results of necropsies are presented. —[*F. C. Jackson, George G. Wright, and James Armstrong: Immunization of Cattle Against Experimental Anthrax with Alum-Precipitated Protective Antigen or Spore Vaccine. Am. J. Vet. Res., 18, (Oct., 1957): 771-777.*]

Infectious Bronchitis Vaccines in Chickens

A study of the relationships between serum-neutralization (SN) titers and responses to intratracheal challenge of birds vaccinated with three strains of infectious bronchitis virus (IBV) showed that a direct correlation between them existed in only one of the three vaccines tested.

Noticeable discrepancies between the SN titers and the challenge results were observed in birds vaccinated with the other two strains of IBV.—[*L. G. Raggi and G. G. Lee: Relationship Between Immunity and Serum-Neutralization Titers in Chickens Inoculated with Three Infectious Bronchitis Vaccines. Am. J. Vet. Res., 18, (Oct., 1957): 740-742.*]

Skin Glands of the Dog

Subgross and microscopic observations were made of hair follicles, epidermis, dermis, and skin glands of 58 dogs. The morphological features are described and illustrated. Four areas were studied in detail—the planum nasale, foot pad, hairy skin, and specialized tail gland area. The development of the simple hair follicle at birth to a compound follicle at maturity is described, and the presence of epidermal papillae on the hairy skin is discussed. The existence of a specialized cutaneous gland area on the dorsal aspect of the tail of domestic Canidae, characterized by large sebaceous and apocrine glands related to simple hair follicles, suggests a similarity to those reported in wild Canidae.—[*James E. Lovell and Robert Getty: The Hair Follicle, Epidermis, Dermis, and Skin Glands of the Dog. Am. J. Vet. Res., 18, (Oct., 1957): 873-885.*]

BOOKS AND REPORTS

The Inheritance of Coat Color in Dogs

This book summarizes present knowledge of the genetics of coat colors in dogs and gives an analysis and comparison of data derived from the author's own experiments and observations, breeders' records, and correspondence on extreme variations. It consists of three main parts: materials and methods, a technical discussion of genetic data, and an analysis by breed. The study is specifically directed toward geneticists and dog breeders who are interested in the scientific aspects of color types.—[*The Inheritance of Coat Color in Dogs. By Clarence C. Little. Comstock Publishing Associates, 124 Roberts Place, Ithaca, N.Y. 1957. Price $4.00.*]

Guide to Medical Writing

This practical manual provides basic information for manuscript preparation—particularly in medical and related fields. It tells how to organize, properly title, write concisely, and summarize an article; also how to capture reader interest in the opening paragraph, and how to search literature and set up a reference list. There is information on illustrations—how to prepare and submit them and how to write legends. Copy revision and proofreading procedures are discussed and there are chapters on how to prepare abstracts and book reviews. For the writer requiring fundamentals, there is information on capitalization, spelling, and abbreviations; how to deal with names, numbers, and nomenclature. This is an excellent guide for anyone who plans to contribute to medical or veterinary medical, literature.—[*Guide to Medical Writing. By Henry A. Davidson. 338 pages. Ronald Press Co., 15 East 26th St., New York 10, N.Y. 1957. Price $5.00.*]

THE NEWS

Dr. Comar Appointed Director of Radiation Research Unit at Cornell University

Dr. Cyril Comar has been appointed director of a research program relating to the hazards of radiation fall-out to farm animals, especially milk-producing animals, and the secondary hazards to man resulting from consumption of products of these animals, at the New York state Veterinary College, Cornell University. A course in radiation biology will be offered during the spring semester and annually thereafter to a group of advanced undergraduate and graduate students.

Dr. Comar, a biochemist, was research associate in chemistry at the Michigan State University in 1941-1943; professor of biochemistry at the University of Florida, 1943-1948; professor of biophysics and laboratory director, University of Tennessee, 1948-1954, and chief of biomedical research, Oak Ridge Institute of Nuclear Studies, U. S. Atomic Energy Commission, 1954-1957.

Dr. Cyril Comar

Of the 88 articles Dr. Comar has published on biochemical and biophysical subjects, all but the first 15 are related to radiation. He is a member of the American Chemical Society, American Institute of Nutrition, American Institute of Biological Chemists, Society of Animal Production, Society for Experimental Biology and Medicine. In 1956, he was elected to honorary membership in the AVMA for his work on nutrition and physiology of domestic animals and his interest in the training of veterinarians at the Oak Ridge Institute.

Dr. Payne Joins Department of Animal Pathology, University of Nebraska

Dr. Loyal C. Payne has been appointed associate professor of animal pathology and associate physiologist in the Department of Animal Pathology and Hygiene, College of Agriculture, University of Nebraska, at Lincoln. Dr. Payne received his D.V.M. from Kansas State College in 1941, and spent the following year in the Department of Physiology, New York State Veterinary College, Cornell University. He was a member of the staff of Iowa State College from 1942 until 1957, receiving the M.S. degree in 1947 and Ph.D. degree in 1953 from that institution.

Dr. Loyal C. Payne

He is a member of the American Veterinary Medical Association, Sigma Xi, Gamma Sigma Delta, Phi Kappa Phi, the National Board of Veterinary Medical Examiners, and the Society of Animal Production.

His duties at the University of Nebraska will include the teaching of veterinary anatomy and physiology to preveterinary agricultural students and to graduate students minoring in animal pathology. His research projects will be concerned primarily with the physiology of swine and beef cattle.

Enrollment in Veterinary Schools 1957-1958

The number of students enrolled in the 17 schools of veterinary medicine in the United States and two in Canada for the present year is the largest for any year for which records are available.

Enrollment in 1957-1958 is 1,627 greater than it was 20 years ago (1937-1938). Of this increase, 1,129 are enrolled in schools established since 1938. Schools that were in operation 20 years ago have increased their enrollment by 498 (table 1).

Comparing 1947-1948 with the present shows a gain of 996 in total enrollment. The 12 schools established prior to 1938 contributed 98 of this total.

Comparing 1956-1957 with the present enrollment shows an overall gain of 43 students. The 1,037 first-year students in the 19 schools is 14 larger, the second year class is smaller by 15, the third year is larger by 30, and the fourth year class is 74 students larger than last year's senior class (table 2).

TABLE 1—Student Enrollment by Decades 1937-1957

	1937-38	1947-48	1957-58
Alabama Polytechnic Institute	157	266	232
Colorado State University	130	199	261
Cornell University	153	143	206
Iowa State College	212	211	264
Kansas State College	220	202	273
Michigan State University	203	228	233
Ohio State University	221	235	274
Pennsylvania, University of	176	169	191
Quebec Veterinary School	53	55	97
Texas A. & M. College	257	266	240
Toronto, University of	241	455	209
Washington, State College of	147	141	188
Subtotal	2,170	2,570	2,668
Georgia, University of		104	242
Minnesota, University of		24	180
Missouri, University of		57	118
Tuskegee Institute		46	92
Subtotal		231	632
California, University of			200
Illinois, University of			143
Oklahoma State University			154
Subtotal			497
TOTAL	2,170	2,801	3,797

The number of veterinarians engaged in graduate study now, ten years ago, and 20 years ago (1938) is shown in table 3.

The number of graduate students (with D.V.M. degrees) in 1957-1958 is 53 greater than last year. This is the largest increase in any single year (181 to 234).

Of the 3,797 undergraduate students, 95 are women. The class distribution of women is as follows: 28 freshmen, 21 sophomores, 16 juniors, and 30 seniors.

TABLE 3—Veterinarians Engaged in Graduate Study

	1937-38	1948-49*	1957-58
Alabama Polytechnic Institute	4	2	7
California, University of	—	—	11
Colorado State University	0	2	10
Cornell University	12	15	21
Georgia, University of	—	0	6
Illinois, University of	—	—	19
Iowa State College	6	9	18
Kansas State College	1	11	7
Michigan State University	7	35	40
Minnesota, University of	—	0	24
Missouri, University of	—	0	4
Ohio State University	6	10	24
Oklahoma State University	—	—	7
Pennsylvania, University of	0	0	7
Quebec Veterinary School	0	0	0
Texas A. & M. College	2	1	15
Toronto, University of	0	0	11
Tuskegee Institute	—	0	0
Washington, State College of	0	2	3
TOTAL	38	87	234

*1948-1949 figures are used to indicate number of graduate students ten years ago.

The relative amount of preprofessional training of the 1,037 first-year students is shown in table 4.

TABLE 4—Preprofessional Training of First-Year Students

	2 Years	3 Years	4 Years	5 Years or more
Number	472	286	195	84
Percentage	45.0	27.8	19.0	8.2

The new class contains 231 students who already have bachelor degrees and 11 with master degrees.

Those enrolled were selected from approximately 2,400 applicants—a ratio of 1:2.4, the same as in recent years.

TABLE 2— Veterinary Student Enrollment for the Academic Year 1957-1958

Schools	Fresh.	Soph.	Jun.	Sen.	Total 1957	Total 1956	Change	Spec.	Grad.
Alabama Polytechnic Institute	64	50	54	64	232	235	− 3	0	7
California, University of	52	49	51	48	200	192	+ 8	3	11
Colorado State University	72	60	67	62	261	261	0	0	10
Cornell University	60	48	48	50	206	202	+ 4	0	21
Georgia, University of	62	62	54	64	242	238	+ 4	2	6
Illinois, University of	47	40	26	30	143	140	+ 3	0	19
Iowa State College	72	63	69	60	264	257	+ 7	0	18
Kansas State College	70	69	70	65	274	264	+10	0	7
Michigan State University	64	62	49	58	233	240	− 7	7	40
Minnesota, University of	47	48	43	42	180	185	− 5	0	24
Missouri, University of	30	29	30	29	118	118	0	0	4
Ohio State University	72	66	68	67	273	283	−10	0	24
Oklahoma State University	42	40	37	35	154	148	+ 6	2	7
Pennsylvania, University of	55	47	43	46	191	194	− 3	0	7
Quebec Veterinary School*	19	33	22	23	97	97	0	0	0
Texas A. & M. College	64	57	56	63	240	232	+ 8	6	15
Toronto, University of*	60	45	52	52	209	202	+ 7	0	11
Tuskegee Institute	35	18	19	20	92	82	+10	0	0
Washington, State College of	50	49	41	48	188	184	+ 4	1	3
TOTAL	1,037	935	899	926	3,797	3,754	+43	21	234

*First year not included.

American Board of Veterinary Toxicologists Established

A meeting of the committee of veterinary toxicologists was held on Sept. 11, 1957, in Longview, Wash., in connection with the annual meeting of the Washington State V.M.A. The result of the session was the formalizing of the group under the name of the "American Board of Veterinary Toxicologists," the broadening of its scope, and the clarification of its aims and objectives.

Dr. H. E. Furguson, Anaconda, Mont., was named president and Dr. G. R. Spencer, State College of Washington, Pullman, vice-president. A committee headed by Dr. Furguson is working on a constitution and bylaws for the consideration of the board at its next meeting which will be held in Salt Lake City, Utah, Jan. 15, 1958, in connection with the Intermountain Veterinary Medical Association meeting, January 16-18.

One of the first projects of the board will be to prepare a 35-mm. film slide series on fluorosis, which will be made available around the first of the year through the AVMA film library and the board's secretary.

Information concerning the board and its requirements for membership may be obtained by writing to Dr. William F. Harris, secretary, 1102 East Main St., Puyallup, Wash.

American Association of Equine Practitioners Convention

The American Association of Equine Practitioners will hold its 1957 convention on December 16-17, at the LaSalle Hotel, Chicago, Ill.

The speakers and their subjects scheduled for the first day of the meeting are: Drs. David K. Detweiler, University of Pennsylvania, Philadelphia—Examination of the Equine Heart; M. B. Teigland, practitioner, Opa Locka, Fla.—Studies on Equine Infectious Anemia; Delano L. Proctor, Jr.—practitioner, Lexington, Ky.—Surgical Treatment of Tendinitis.

Dr. W. W. Armistead, president of the AVMA and dean, College of Veterinary Medicine, Michigan State University, will be the speaker at the association's dinner on December 16.

Among the veterinarians to speak on the second day of the convention are: Drs. William O. Reed, practitioner, Elmont, N.Y.—Ether Anesthesia in the Horse; Nolen D. Connors, Upjohn's Veterinary Experiment Station, Richland, Mich.—The Safety of Piperazine Carbon—Disulfide Complex in Foals, Weanlings, and Broodmares; and J. H. Drudge, Department of Animal Pathology, University of Kentucky, Lexington—Critical Tests and Field Studies with Parvex Against Parasites of the Horse.

Mr. Marshall Cassidy, vice-president and director of racing, Greater New York Association, and executive secretary, New York Jockey Club, will discuss the correlation of interests common to race track management, horsemen, official chemists, and veterinary practitioners for the best interests of racing.

Each member present will be asked to submit, anonymously, worthwhile observations and findings on diagnosis or therapy that could be of use to others in the field.

Group Conference—American Boards of Veterinary Medical Examiners

The American Boards of Veterinary Medical Examiners met Aug. 19, 1957, in the Hotel Cleveland, during the AVMA Ninety-Fourth Annual Meeting in Cleveland. Designated representatives from 19 states were present; also there were several alternates from state boards as well as some visitors.

Mr. Charles B. Frasher, personnel consultant for Professional Examination Service, addressed the meeting on the "Current Use of the National Board P.E.S. Examinations."

A total of 600 examinees took the examination this year, Mr. Frasher advised. This represented 14 states and the Canadian Veterinary Medical Association. Three of the states used the examination twice in 1957. Several more states are seriously considering this type of examination next year.

The committee on constitution and by-laws, consisting of Mr. James O. Hoyle, chairman, Drs. Jean V. Smith, and Joseph A. S. Millar, reported and presented a well-drafted proposed constitution and bylaws. This draft was studied and thoroughly discussed, article by article and section by section. The delegates approved it, but moved that the revised draft be presented to each state board for their study as a complete body and their decision reported at the next meeting.

s/CHARLES W. BOWER, *Chairman.*

AMONG THE STATES AND PROVINCES

Idaho

Idaho Southwest Association.—The Idaho Southwest V.M.A. met at Payette, Sept. 27, 1957, for its regular dinner meeting.

The following speakers presented reports: Drs. Floyd W. Frank, Laramie, Wyo.—Vibriosis; Arthur P. Schneider, Boise—Anthrax; Craig Rowan, Weiser—Highlights of the National Convention; and Raymond C. Derrer, Meridian—The Drug Committee's Report.

Dr. Millard W. Ickes, Nampa, president, moderated the discussions which followed each report.

s/MICHAEL INVERSO, *Secretary.*

Illinois

Women's Auxiliary.—On Sept. 18, 1957, 26 members of the Women's Auxiliary to the Northern Illinois V.M.A. met at the Women's Club Food Shop, Rockford, Ill.

At the business meeting, Mrs. E. C. Hannon, Maple Park, president, introduced the following officers of the Auxiliary: Mrs. John Nelson, De Kalb, president-elect; Mrs. D. W. Rawson, Milledgeville, secretary-treasurer-elect; Mrs. C. M. Rodgers, Blandinsville, membership secretary of the AVMA Auxiliary; Mrs. C. W. Larsen, Shabbona, state secretary; and Mrs. R. J. Cyrog, Skokie, state membership secretary.

The members voted to send Mrs. Lewis A. Dykstra, Aurora, and Mrs. James G. Hardenbergh, Rockton, as delegates and Mrs. Edward C. Saunders, Elgin, as alternate, to the annual convention of the AVMA Women's Auxiliary in Philadelphia, in 1958.

Mrs. Hannon presented an interesting resumé of the annual national meeting at Cleveland and Mrs. Rodgers reported on the portions of the meeting that Mrs. Hannon was unable to attend.

s/MRS. D. W. RAWSON, *Secretary-Treasurer.*

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Chicago Association.—The regular monthly meeting of the Chicago V.M.A. was held in the Lincoln Room of the LaSalle Hotel, Nov. 12, 1957.

Dr. John R. Dick, Fort Dodge Laboratories, spoke on "Canine Leptospirosis" and presented demonstrations of serological tests.

s/MARK E. DAVENPORT, *Secretary.*

Iowa

Midwest Small Animal and American Animal Hospital Associations.—The twentieth annual meeting of the Midwest Small Animal Association and the regional meeting of the American Animal Hospital Association met at the Hotel Burlington, Burlington, Iowa, Nov. 13-14, 1957.

Among the speakers and their subjects on the program were: Drs. R. M. Schwartzman, Morris Foundation Research Fellow, University of Minnesota, St. Paul—Canine Dermatology; George Eberhart, Kansas State College, Manhattan—Public Relations; Richard W. Storey, practitioner, Muskegon, Mich.—Canine Distemper Immunization; Kenneth B. Hass, Upjohn Co., Kalamazoo, Mich.—Newer Therapeutic Agents; and Glen L. Dunlap, Haver-Lockhart Laboratories, Kansas City, Mo.—Rabies Vaccination.

A panel discussion closed each day of the meeting.

Kansas

Kansas City Association.—The regular October meeting of the Kansas City V.M.A. was held at the Hotel President, Kansas City, Mo., Oct. 17, 1957.

The program consisted of talks by two outstanding clinicians. Dr. B. W. Kingrey, director, clinical medicine and surgery, Iowa State College, Ames, spoke on the improved techniques in surgery, using graphic illustration from a color film.

Dr. D. L. Baker, small animal clinician, Ames, Iowa, presented an audiovisual demonstration and lecture on abnormal heart sounds and their diagnostic interpretation.

s/R. E. GUILFOIL, *Secretary-Treasurer.*

Louisiana

Louisiana Association.—The sixth annual fall conference of the Louisiana V.M.A. met Aug. 28-29, 1957, at the Jung Hotel in New Orleans. This meeting attracted veterinarians from Louisiana, Texas, and Mississippi.

Among the speakers on the program were: Drs. F. G. Fielder, Schering Corporation, Bloomfield, N. J.; C. T. Mason, practitioner, Little Rock, Ark.; C. W. Schulz, head, Department of Veterinary Medicine and Surgery, Ohio State University, Columbus; B. F. Hoerlein, Alabama Polytechnic Institute, Auburn; and E. E. Saulmon, assistant chief, ARS, Washington, D. C.

The new officers elected for the coming year are: Drs. W. H. Lockard, Zachary, president; J. N. Thomas, Welsh, vice-president; O. C. Granzin, Baton Rouge, treasurer; and A. M. Stefanski, Crowley, secretary.

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Women's Auxiliary.—The Women's Auxiliary to the Louisiana V.M.A. met on Aug. 28-29, 1957, in New Orleans, in conjunction with the sixth annual meeting of the veterinary association. Mrs. J. E. Morrison, Opelousas, presided over the assembly.

Michigan

A Citizens' Advisory Committee to the Michigan V.M.A. Appointed.—The formation of a citizens' advisory committee to the Michigan Veterinary Medical Association was announced Oct. 11, 1957, by Dr. Stephen R. Kelly, president.

Members of the committee, the first such laymen's advisory council in the 75-year history of the Michigan V.M.A., are: Keith Middleton, Lake Orion, dairy farmer; S. P. Clickner, Detroit, breeder of show dogs; and Clifton Johnson, managing director, Michigan Humane Society.

The committee will provide the layman's point of view on matters of veterinary medical practice in the care of large animals, small animals, and in the public service field.

"We believe the availability of lay counsel and guidance will contribute substantially to a better understanding between the veterinarian and his clients. As with human medicine, the

practice of veterinary medicine has become increasingly complex due to the development of new drugs and surgical techniques. We feel there is a vital need for closer contact with the public to insure a continuing high level of professional service," Dr. Kelly said.

There are now more than 600 veterinarians engaged in large and small animal practice and in public health service in Michigan.

s/ROBERT F. WILLSON, *Executive Secretary.*

New Jersey

Southern New Jersey Association.—The officers elected by the Southern New Jersey V.M.A. for the year 1957-1958 are: Drs. O. K. Fox, Moorestown, president; F. W. Wolfe, Salem, vice-president; R. M. Sauer, Westmont, secretary; and F. J. Olbrich, Blackwood, treasurer.

s/R. M. SAUER, *Secretary.*

New York

Dr. Robert S. MacKellar, Sr., Honored.—At the thirteenth annual canine cavalcade held in Rockefeller Center, N.Y., Sept. 19, 1957, Brig. Gen. Wayne O. Kester, retired chief, U.S.A.F. (V.C.), presented an award, saluting the veterinary profession's contributions to the welfare of dogs, to Dr. Robert S. Mac Kellar, Sr., oldest and still practicing veterinarian in New York City.

Dr. Robert S. MacKellar (right), New York City, accepting the New York City V.M.A. Award, recognizing his 63 years of service, from Brig. Gen. Wayne O. Kester (Ret.).

This award pays special tribute to the work of veterinarians engaged in the maintenance and preservation of the good health of all dogs.

• • •

State Association.—The sixty-sixth annual meeting of the New York State V.M.A. convened Sept. 10-13, 1957, at the Hotel Statler, Buffalo, N.Y.

Some of the speakers and their subjects included in the four-day program were: Drs. Fred J. Kingma, Washington, D.C.—Some Criteria for Optimal Anesthesia; Wayne H. Riser, Skokie, Ill.—X-ray Interpretation and Hospital Standards; Wade O. Brinker, East Lansing, Mich.—Preoperative and Postoperative Treatment of Fractures in Small Animals; Elliot A. Maynard, Rochester, N.Y.—Antibiotics in Milk; Richard S. Guthrie, Ithaca, N.Y. —Inclusion of Dyes in Mastitis Preparations; C. Seymour Card, Jr., Augusta, Maine—Nutrition Problems in the Dairy Herd; and John B. Herrick, Ames, Iowa—Bovine Infertility and the Practitioner.

On the last day of the meeting, the following three seminars were held: Small Animals—The Acute Abdomen in Small Animals; Horse Practice—Treatment of Common Lameness; Cattle Practice—Diseases of the Dairy Herd. All the speakers who participated in the program also took part in the question-and-answer luncheon which followed the seminars.

At the business session of the meeting, the membership voted to employ a full-time professional executive secretary. The officers elected for the ensuing year are: Drs. James D. Sweet, Chateaugay, president; Peter Olafson, Ithaca, president-elect; William J. Sellman, Utica, treasurer.

Next year, the New York State V.M.A. will hold their annual convention at the Concord Hotel, Kiamesha Lake, N.Y., Sept. 10-12, 1958.

s/JOAN S. HALAT, *Executive Secretary.*

Quebec

Annual Convention of the V.M.A. of the Province of Quebec.—The annual convention of the Province of Quebec V.M.A. was held at the School of Veterinary Medicine of Saint-Hyacinth, Oct. 9, 1957. Dr. Ephrem Jacques, assistant director of the school, presided.

The following lectures were presented by the faculty of the veterinary school: Drs. J. D. Nadeau—Enterotoxemia; Maurice Panisset—Complex Mucosal Diseases; Roland Filion—Milker and Mammitis.

The officers elected for the year 1957-1958 are: Drs. Paul Marois, laboratory head, Institute of Microbiology and Hygiene, University

of Montreal, president; J. D. Nadeau, controller-administrator of the school, first vice-president; and Ephrem Jacques, assistant director of the school, second vice-president.

s/JACQUES SAINT-GEORGES, *Secretary.*

Wisconsin

Milwaukee Association.—At the regularly scheduled October, 1957, meeting of the Milwaukee V.M.A., Dr. Berstein, Glencoe, Ill., and Dr. Riser, Skokie, Ill., presented informative dissertations on hip dysplasia and allied disorders.

Thirty-one members attended the meeting.

s/R. H. STEINKRAUS, *Secretary.*

FOREIGN NEWS

Italy

Death of Professor Ascoli.—Professor Alberto Ascoli, M.D., former professor in the veterinary school at Milan, died on Sept. 28, 1957, of a heart attack at the age of 80.

Professor Ascoli fled to the United States when World War II broke out and taught bacteriology at Middlesex University veterinary school. Later, he taught at Rutgers and at New York University. He was a well-known authority on tuberculosis, developed a B.C.G. vaccine almost simultaneously with a similar vaccine prepared by the Pasteur Institute in Paris, and was at one time director of the Italian Vaccine Institute.

Uruguay

Uruguay Association.—The Veterinary Medical Association of Uruguay celebrated its fiftieth anniversary on May 10, 1957, in conjunction with the Second National Veterinary Congress held in Montevideo, May 6-10, 1957.

Dr. G. Lockhart presided over the assembly which included representatives from Argentina, Brazil, and Chile.

STATE BOARD EXAMINATIONS

CALIFORNIA—January 30-31 and Feb. 1, 1958. Davis. William E. Barbeau, executive secretary, 1020 N. St., Sacramento 14, Calif.

COLORADO—Jan. 15-16, 1958, Arvada; and first week in June, depending on graduation date, Fort Collins. W. D. Stauffer, secretary, 5500 Wadsworth Blvd., Arvada, Colo.

ILLINOIS—Dec. 4-6, 1957, Chicago. F. B. Selcke, registration superintendent, Springfield, Ill.

MINNESOTA—Jan. 6-7, 1958, St. Paul. A. C. Spannaus, secretary, Route 1, Waconia, Minn.

NEW JERSEY—Dec. 27-28, 1957, Trenton. Joseph A. S. Millar, secretary, Box 172, Deal, N.J.

NEW YORK—Dec. 10-13, 1957. Written examination, New York, Albany, Syracuse, and Buffalo. James O. Hoyle, secretary, 23 Pearl St., Albany, N. Y.

NORTH DAKOTA—April 9-10, 1958, Fargo. M. C. Hawn, secretary-treasurer, 1407 13 St. N., Fargo, N. Dak.

NEW MEXICO—Jan. 13, 1958, Santa Fe. Edwin J. Smith, secretary, P.O. Box 4385, Santa Fe, N.M.

PUERTO RICO—Jan. 7, 1958. San Juan. Joaquin Mercado Cruz, secretary, Box 3271, San Juan, P. R.

TEXAS—Jan. 15-17, 1958, Austin. T. D. Weaver, executive secretary, 207 Capital National Bank Building, Austin 16, Texas.

DEATHS

Star indicates member of AVMA

★**T. H. Ferguson** (OVC '96), 84, former AVMA president and widely known veterinarian of Lake Geneva, Wis., died of a heart attack at his home there, Oct. 12, 1957.

Born in 1873, at Linn, Wis., Dr. Ferguson attended Lake Geneva high school before enrolling at Ontario Veterinary College. Following graduation, he began practice in Lake Geneva, his grandparents having been among the first settlers in that area.

He became recognized as an authority in bovine medicine and surgery and was called upon to lecture and give demonstrations before veterinary audiences in many parts of the country. He was also active in civic affairs and a highly respected leader in his community and state. For many years, he was a member of the Wisconsin Board of Veterinary Examiners and served either as president or secretary for much of that time.

In professional association work, Dr. Ferguson was also a leader and the recipient of many offices and honors. He served as president of the Wisconsin State V.M.A. and was continually active in its work. He served the AVMA in several capacities: as a member and chairman of the Committee on Education, as section officer, vice-president, and president (1929-1930).

In 1938, he was one of nine men who received gold keys in recognition of outstanding services to the Association and, in 1946, he was awarded the Twelfth International Veterinary Congress Prize by the Association for his accomplishments in veterinary science.

Surviving are his three sons, Dr. Stanley E. Ferguson, Lake Geneva, a veterinarian; Dr. W. Richard Ferguson, Baltimore, a physician; and John H. Ferguson, Wood, Wis.; two daughters; and eight grandchildren.

Elmer C. Johnson (KVC '07), 75, Mankato, Kan., died Aug. 28, 1957, after a long illness.

Dr. Johnson practiced in Jewell County, Kansas, for 50 years before he retired about a year ago because of ill health.

He is survived by his widow.

Francis E. Ludgate (CVC '11), 72, Fort Dodge, Iowa, died Aug. 16, 1957, after a long illness.

B. O. Moore (ONT '15), 64, Harrison, Ark., died Sept. 1, 1957, in the Boone County Hospital, after a heart attack.

Dr. Moore, a native of Tennessee, had moved from Reno, Nev., to Harrison in 1953.

He is survived by two sons, Paul L. and Benjamin O., Jr.; two brothers; and one sister.

★**Orin H. Osborn** (MCK '12), 79, Seaside, Ore., died at his home in August, 1957, after a two-year illness.

Dr. Osborn was born near Cooksville, Wis., and as a child moved with his parents by covered wagon to Marshalltown, then to Farnhamville, Iowa. Eight years prior to his retirement, Dr. Osborn owned and operated a mink farm. He had lived in Seaside since 1948.

He was a member of the Minnesota State Veterinary Medical Society and the Paynesville (Minn.) Masonic Lodge No. 71, A.F. & A.M. He is survived by his widow, Myrtle, a daughter, and two brothers.

Frank H. Riester (IND '01), 79, Bueshel, Ky., died Aug. 19, 1957.

Dr. Riester, who retired from veterinary practice five years ago, was the first man to conduct saliva tests in Kentucky Thoroughbred race horses.

He was president of the Kentucky V.M.A. in 1930-1939 and he served as secretary-treasurer of the Association for eight years from 1937 to 1945.

Dr. Riester is survived by his widow, Mary McCory Riester, a brother, and two sisters.

★**Jack K. Skiles** (KCV '16), 66, Denton, Texas, died Sept. 4, 1957, in Flow Memorial Hospital, after a two-year illness.

In Denton County, Dr. Skiles was recognized as a successful farmer, dairyman, and cattleman, as well as a practicing veterinarian.

He was the organizer of the Denton County Fair and served as its president, manager, and director for nearly three decades. At the time of his death, he was honorary president of the fair association.

Dr. Skiles was active in the Kiwanis Club and was past-president of the Kiwanis in Denton. He was also a member of the Masonic Lodge, Shriners, and the Moslah Temple, Fort Worth.

Among many other activities, Dr. Skiles served as a captain in the Army in the first World War.

Dr. Skiles is survived by two sisters, two brothers, and several nieces and nephews.

★**George W. Staggs** (WSC '20), 64, Tacoma, Wash., died Aug. 18, 1957, following a heart attack while on a weekend fishing trip in eastern Lewis County.

Dr. Staggs had practiced in Tacoma where he owned and operated the Tacoma Veterinary Hospital from 1942 until his retirement last year. He was a past-master of the Chehalis Kiwanis club, past-president of the Washington State V.M.A., and a member of the Adna Grange, Elks Lodge, and the South Puget Sound Medical Association.

He is survived by his widow, Martha Alma Hann Staggs, a son, and a daughter.

★**Fred J. Standford** (MCK '08), 81, Fayetteville, Ark., died at his home Sept. 8, 1957, after a lingering illness.

Dr. Standford served as the first state veterinarian in Arkansas. He was a charter member of the Arkansas V.M.A., a member of the Royal Arch Masons, Washington Lodge No. 1, and the Eastern Star.

From 1912 to 1922, Dr. Standford operated the Arkansas Veterinary College in Fayetteville.

He is survived by two sons, three daughters, and two brothers.

★**Sidney A. Steiner** (UP '36), 54, Philadelphia, Pa., died at the University Hospital there, Sept. 1, 1957.

Dr. Steiner had been associated with the Agricultural Research Service of the U. S. Department of Agriculture for 20 years. He held a master's degree in pathology.

Among Dr. Steiner's activities were the Keystone Veterinary Medical Society, AVMA, and the Pannonia Beneficial Association. He is survived by his widow, Rose, a son, and a daughter.

Leon J. Sutton (ISC '54), 34, Annandale, N. J., died Sept. 3, 1957, in Hannemann Hospital, Philadelphia.

Dr. Sutton had been employed by the State of New Jersey for several years as an inspector of dairy and other animals. He is survived by his widow, Dorothy Purdy Sutton, and a daughter.

William M. Thorning (WSC '41), 41, Hollister, Calif., died from a heart attack July 25, 1957.

Delmar C. West (KVC '14), 74, Milford, Neb., died Aug. 27, 1957. Dr. West was a lifelong resident of Seward County where his parents were the first white settlers. He was a member of the school board, town board, chamber of commerce, and the volunteer fire department.

Surviving are his daughter, two sisters, and a grandson.

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Because of an erroneous report received from the U.S. Post Office, the JOURNAL reported the death of Augustus S. Martin (KCV '17) in the Oct. 1, 1957, issue. Our apologies to Dr. Martin.—The Editors.

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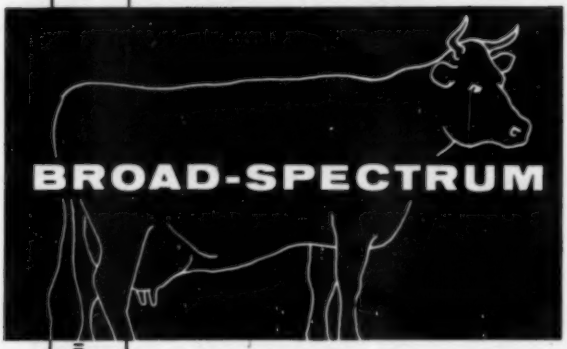

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APPLICATIONS

Applicants—Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. This certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative Bylaws.

ELLIS, EDWIN M.
Coastal Plain Experimental Station, Tifton, Ga.
D.V.M., Michigan State University, 1956.

ESTRADA, EMILIO
63 Elm St., West Haven, Conn.
D.V.M., University of Montreal, 1944.

PAPP, EUGENE
173 Wayne St., Hilliards, Ohio
D.V.M., Hungarian University of Agri. Science, 1943.

SPEER, EDWARD W.
Stanwood, Iowa.
D.V.M., Kansas State College, 1952.

Applicants — Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and names of his endorsers.

Second Listing

MUNOZ, ARMANDO, D.V.M., Casilla No. 3985, Guayaquil, Ecuador.

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

First Listing

University of Pennsylvania

ANDERSON, BURLEIGH P., V.M.D.
301 So. Union Ave., Havre De Grace, Md.
Vouchers: James H. Mark and Donald G. Lee.

CROASMUN, WILLIAM A., V.M.D.
R.D. 3, Titusville, Pa.
Vouchers: William C. G. Savage and Melvin Vaclavik.

Tuskegee Institute

DUDLEY, WILLIAM N., D.V.M.
962 Canal Ave., St. Paul, Minn.
Vouchers: G. W. Cooper and Edward T. Braye.

Ontario Veterinary College

SACK, WOLFGANG, D.V.M.
6448 N. Clark St., Chicago, Ill.
Vouchers: J. H. Ballantyne and H. J. Neely.

Second Listing

Colorado State University

JONES, WILLIAM L., D.V.M., Rt. 2, Box 29, Fort Collins, Colo.

University of Pennsylvania

LEMOS, EDWARD J., V.M.D., Horseneck Rd., Dartmouth, Mass.

Tuskegee Institute

SMITH, LORRAINE E., D.V.M., 4123 - 4th St., N.W., Washington, D.C.

DAWKINS, WHIT M., D.V.M., 326 W. 19th, South Sioux City, Neb.

AVMA Research Fellowships Available

The Research Council of the AVMA announces the availability of a number of fellowships for postgraduate training for the academic year, 1958-1959.

The recipient of a fellowship must be a veterinarian and a citizen of the United States or Canada. Veterinary students who expect to graduate at the end of the current school year and who wish to follow a career in research may apply for a fellowship.

The latest date for filing the completed application is Feb. 15, 1958. Approximately one month is required for processing completed applications after receipt by the secretary of the Council. Qualified persons should secure and submit applications as early as possible to insure their file being complete for presentation to the Committee on Fellowships.

The Committee on Fellowships of the Research Council will meet in March to consider applications, and the awards will be announced soon afterward. The stipend will be determined in each case by the needs of the individual, the location of the school in which he proposes to work, and other factors. In general, the stipends range from $100 monthly and upward.

Any qualified person interested in graduate training may obtain application blanks and other information by writing to Dr. C. H. Cunningham, secretary, AVMA Research Council, Department of Microbiology and Public Health, College of Veterinary Medicine, Michigan State University, East Lansing, Mich.

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International Conference on Livestock Stress

The world's first concerted scientific effort to determine the effects of stress on the health, weight, and meat quality of livestock, and to seek new methods of preventing such stress, will be made at Kansas City, Mo., on Dec. 10, 1957, when leading veterinary authorities from many states and several foreign countries will gather at the Muehlbach Hotel, Kansas City, Mo., for the First International Research Conference on the Effects of Stress on Livestock. The conference is sponsored by Jensen-Salsbery Laboratories of Kansas City, largest pharmaceutical house in the U. S. devoted solely to veterinary practice.

Dr. A. H. Quin

Dr. Carl A. Brandly

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Quart bottle	2.95
Pint bottle	1.65
8 fl. oz. bottle	.92
4 fl. oz. bottle	.54

#684, 'PARLAMATE' Citrate Tablets, 500 mg. (Each tablet contains the equivalent of 200 mg. available piperazine.)

Bottle of 1,000	$20.00
Bottle of 500	10.67
Bottle of 100	2.67

#680, 'PARLAMATE' Citrate Powder. (Contains 100% piperazine citrate.) In pre-weighed dispensing packages convenient for office and field use.

100 lb. bulk, per lb.	$4.73
50 lb. bulk, per lb.	4.80
25 lb. bulk, per lb.	4.87
10 lb. bulk, per lb.	4.93
5 lb. bulk, per lb.	4.97
16 oz. bottle	$5.00
8 oz. bottle	3.00
4 oz. bottle	1.67
1 oz. bottle	.50

For Puppies, Cats and other Small Animal Use:

#681, 'PARLAMATE' Citrate Tablets, 250 mg. (Each tablet contains the equivalent of 100 mg. available piperazine.)

Bottle of 1,000	$10.00
Bottle of 500	5.34
Bottle of 100	1.34

#682, 'PARLAMATE' Citrate Liquid 5%. (Each teaspoonful, approximately 5 cc., supplies 250 mg. piperazine citrate, which is equivalent to 100 mg. available piperazine.)

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Pint bottle	1.34

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been recognized for some time. In addition, stress is believed to be the principal cause of weight loss and injuries in shipment and of what is known as the "dark cutter syndrome", a condition which affects the quality of meat, and also that it may result in incomplete drainage of blood after slaughtering.

The combined economic loss to livestock raisers, packers, and consumers is close to a billion dollars, according to Ray H. Cuff, Kansas City livestock commissioner, an authority on livestock shipping and handling.

Among those who will participate in the conference will be Dr. A. H. Quin, former president of the AVMA, chairman; Dr. Carl A. Brandly, dean of the School of Veterinary Medicine, University of Illinois; Dr. Harold D. Hedrick, professor of animal husbandry, University of Missouri; Dr. Nelson B. King, research veterinarian, Ohio State University, Agricultural Experiment Station; Dr. Roy S. Nichols, professor of veterinary physiology, University of Wisconsin; Dr. Alvin B. Hoerlein, School of Veterinary Medicine of the University of Illinois; Dr. S. K. Sinha, formerly of the University of Manila and the University of Pennsylvania, now director of biological research at Jensen-Salsbery Laboratories, Kansas City; Dr. A. R. Haskell, director of pharmacological research at Jensen-Salsbery; Dr. M. D. Sutter, Jensen-Salsbery director of field research, and Mr. Cuff.

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It also publishes the program and abstracts for the annual meeting of the American Society of Parasitologists in a Supplement.

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GEORGE R. LA RUE

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COMING MEETINGS

American Association of Equine Practitioners. Annual meeting. La Salle Hotel, Chicago, Ill., Dec. 16-17, 1957. M. L. Scott, 42 W. Market St., Akron 8, Ohio, executive secretary.

Pennsylvania, University of. Fifty-eighth annual conference of veterinarians. Jan. 7-8, 1958, School of Veterinary Medicine, University of Pennsylvania, Philadelphia. Dr. James Mark, chairman.

Cornell University. Annual conference for veterinarians. New York State Veterinary College, Ithaca, Jan. 8-10, 1958. W. A. Hagan, dean.

Ohio State Veterinary Medical Association. Seventy-fourth annual meeting. Netherland Hilton Hotel, Cincinnati, Jan. 8-10, 1958. Harry C. Sharp, executive secretary.

Oklahoma Veterinary Medical Association. Annual meeting. Hotel Lawtonian, Lawton, Jan. 9-10, 1958. Mrs. Larma Bennett, 2805 S. W. 51 St., Oklahoma City, executive secretary.

Kansas Veterinary Medical Association. Annual convention. Hotel Broadview, Wichita, Jan. 12-14, 1958. K. Maynard Curts, 5236 Delmar Ave., Kansas City 3, Kan., secretary.

Tennessee Veterinary Medical Association. Annual meeting. Hotel Andrew Jackson, Nashville, Jan. 12-14, 1958. H. W. Hayes, 5009 Clinton Pike, Nashville, secretary-treasurer.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Des Moines, Jan. 14-16, 1958. F. B. Young, Waukee, Iowa, secretary.

American Board of Veterinary Toxicologists. Winter meeting. Salt Lake City, Utah, Jan. 15, 1958. William F. Harris, 1102 East Main St., Puyallup, Wash., secretary.

Indiana Veterinary Medical Association. Annual meeting. Hotel Severin, Indianapolis, Ind., Jan. 15-17, 1958. L. M. Borst, 3315 Shelby, Indianapolis, secretary.

Intermountain Veterinary Medical Association. Annual meeting. Hotel Utah, Salt Lake City, Jan. 16-18, 1958. R. A. Bagley, 4600 Creek View Dr., Murray, Utah, secretary.

Minnesota Veterinary Medical Association. Annual meeting. St. Paul Hotel, St. Paul, Jan. 20-22, 1958. B. S. Pomeroy, School of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn., secretary.

Michigan State University. Thirty-fifth annual postgraduate conference for veterinarians, Jan. 22-23, 1958. College of Veterinary Medicine, Michigan State University, East Lansing. W. W. Armistead, dean.

Louisiana State University. Annual veterinary conference. Louisiana State University, Baton Rouge, Jan. 28-29, 1958. W. T. Oglesby, Baton Rouge 3, La., head.

North Carolina State College. Conference for veterinarians. North Carolina State College, Raleigh, Jan. 28-31, 1958. C. D. Grinnells, chairman.

Oregon Veterinary Medical Association. Winter meeting. Portland, Jan. 31-Feb. 1, 1958. Edward L. Holden, P. O. Box 445, Oswego, secretary.

Wisconsin Veterinary Medical Association. Annual meeting. Pfister Hotel, Milwaukee, Wis., Feb. 10-12, 1958. B.A. Beach, 1215 Vilas Ave., Madison, secretary.

Colorado State University. Nineteenth annual veterinary conference. Glover Veterinary Hospital, College of Veterinary Medicine, Colorado State University, Fort Collins, Colo., Feb. 17-19, 1958, Lloyd C. Moss, head Department of Medicine, Colorado State University, secretary.

Illinois State Veterinary Medical Association. Annual convention. LaSalle Hotel, Chicago, Feb. 17-19, 1958. C. B.

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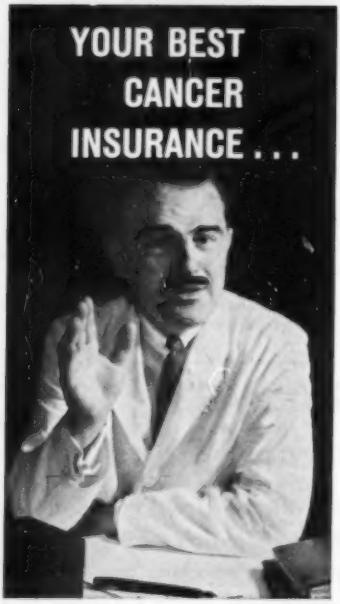

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American Cancer Society

Hostetler, 1385 Whitcomb Ave., Des Plaines, Ill., executive-secretary.

American Animal Hospital Association. Silver anniversary meeting. Drake Hotel, Chicago, Ill., April 23-26, 1958. Joseph A. S. Miller, president.

Oklahoma State University. Oklahoma conference for Veterinarians. College of Veterinary Medicine, Oklahoma State University, Stillwater, May 5-6, 1958. Lester Johnson, Department of Veterinary Medicine and Surgery, chairman.

New York State Veterinary Medical Society. Sixty-seventh annual meeting. Concord Hotel, Kiamesha Lake, N. Y., Sept. 10-12, 1958. Joan S. Halat, 803 Varick St., Utica, N. Y., executive secretary.

Foreign Meetings

Sixth International Congresses on Tropical Medicine and Malaria. Lisbon, Portugal, Sept. 5-13, 1958. Professor Manuel R. Pinto, Institute of Tropical Medicine, Lisbon, secretary-general. (Membership application forms may be obtained by U.S. veterinarians by writing to the AVMA.)

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the second Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, Decatur, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December at 3004 16th St., San Francisco, Calif. Mr. Herb Warren, executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second

Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month, Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. C. Lawson, 40-49 El Camino, Palo Alto, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyea, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, in St. Petersburg. R. L. Brutus, 336 E. 15th St., Hialeah, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

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fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P. O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month, at 6:30 p.m., usually in Cedar Rapids, Iowa. William Rugger, Oxford, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

KANSAS—Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

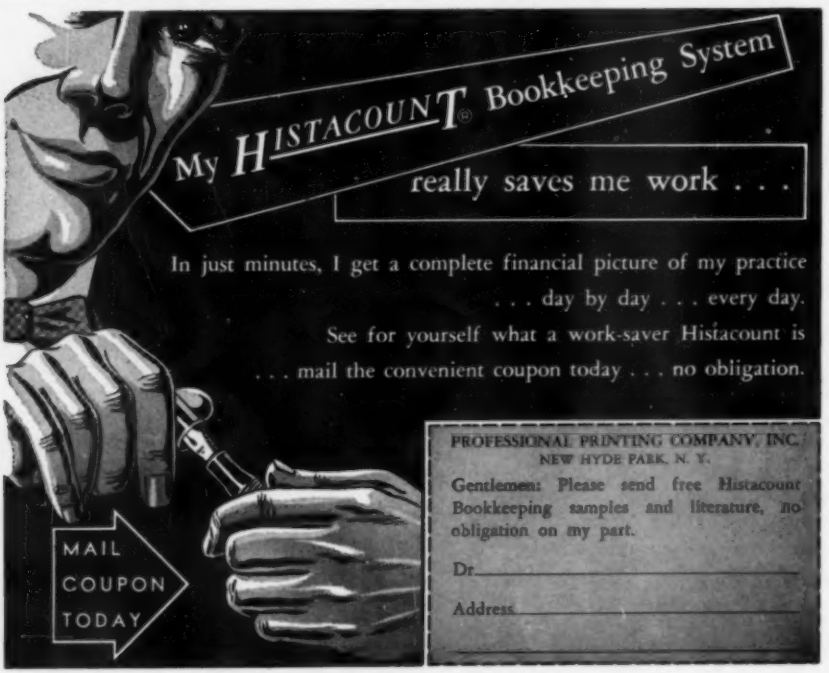
KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Varsailles Rd., Frankford, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Cozrell, Rt. 1, Midland, Mich., secretary.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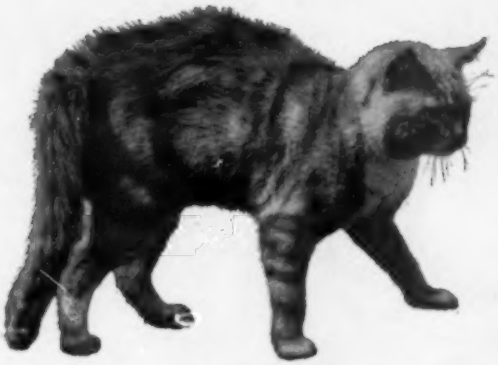

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Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Chester R. Pliegge, 4249 Peck St., St. Louis 7, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Highs Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. R. M. Sauer, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College, Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly, Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Thursday of each month in the Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. James R. Burgess, Rockingham, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

Stark County Veterinary Association, the second Monday of each month. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m., Ireland's Restaurant, Lloyds', 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinarian Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary

PENNSYLVANIA—Del-High Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the Second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

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REFERENCES: 1. Master, J. E.: Vet. M. 50:605 (Nov.) 1955. 2. Belloff, O. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 3. Pollock, S.: J. Am. Vet. M. Ass. 129:274 (Sept.) 1956.

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Figure 1

History.—A 4-year-old male Shepherd dog had been struck by a car seven months before being presented at the hospital. Since the injury, there had been a unilateral (right) nasal discharge. There was no visible wound in the mouth but the right upper canine tooth was missing. A radiograph was made with the dog lying on its back and under anesthesia, the upper jaw pressed against the casette and the mouth held open by means of gauze bandages, one encircling the lower jaw and one the upper jaw.

(Diagnosis and findings are reported on the next page)

Here Is the Diagnosis

(*Continued from preceding page*)

Diagnosis.—Dorso-posteromedian displacement of the right upper canine tooth.

Comment (*C. Lawrence Blakely*).—Surgical exploration revealed that the canine tooth was lying in the nasal cavity, with the root pointed posteriorly and imbedded in the turbinates. We were unable to approach it from the anterior nasal orifice, and it could not be seen through the nostril with any instruments ordinarily available, although one might possibly have seen the apex of the tooth.

Fig. 2—Radiograph (ventrodorsal view) showing the presence of a canine tooth in nasal cavity (arrow).

In approaching it surgically, the lip was elevated and a slightly curved incision was made through the mucous membrane and periosteum of the alveolar ridge of the missing canine tooth, then the lateral and medial alveolar walls of the empty socket were invaded to enter the nasal cavity. With a pair of heavy hemostats, it was possible to force entry into the nasal cavity, seize the tooth, and extract it. The gum and periosteum were then brought back into position and sutured loosely. Recovery was rapid and uncomplicated. This case is reported not because of any great technical difficulty involved, but because of its long standing and rather bizarre nature.

This case was presented by the staff of Angell Memorial Animal Hospital, Boston, Mass.

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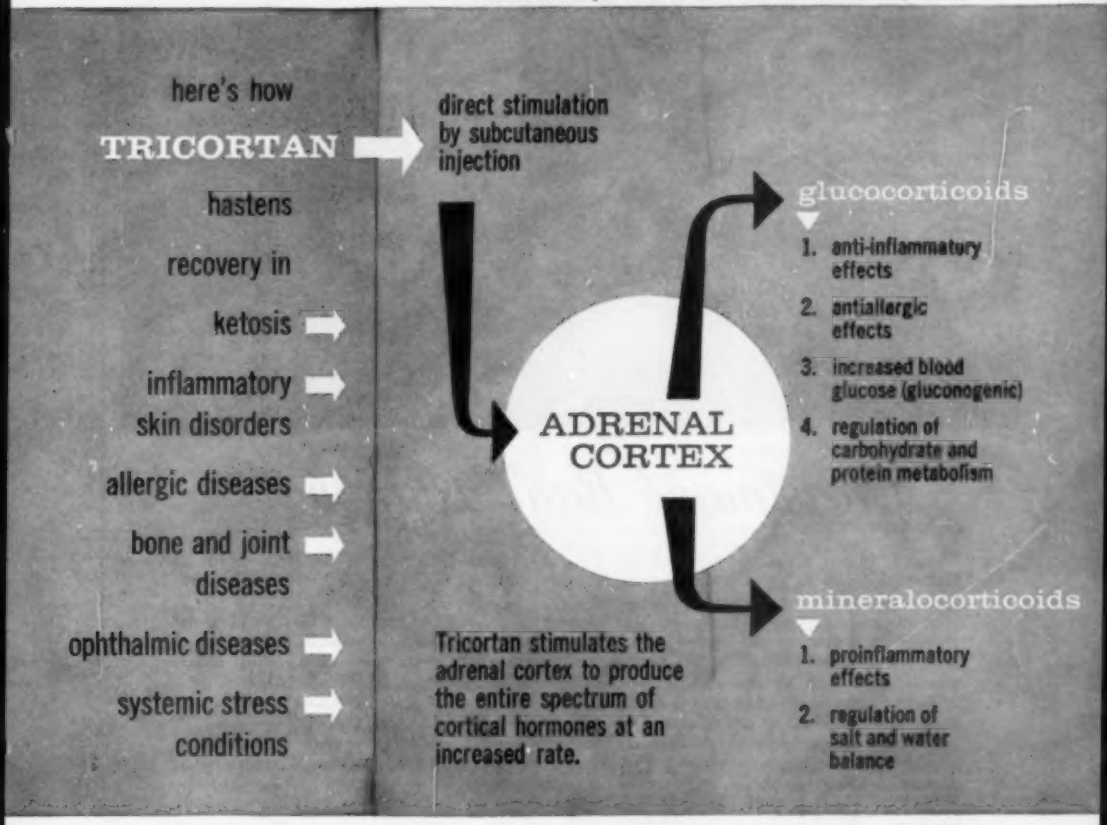

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Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). R. J. O'Hern, P.O. Box 617, Cumberland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

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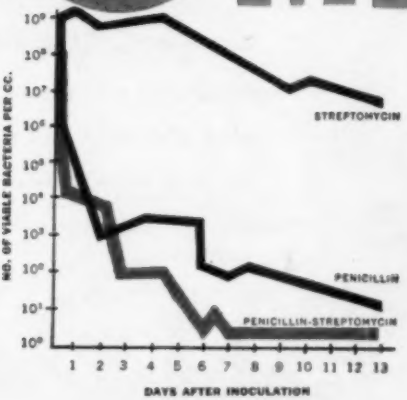

This graph presents the combined, synergistic antibacterial action of penicillin and streptomycin on a culture of *Streptococcus liquefaciens* (strain L. W.).[2]

1. Welch, H.: Antibiotic Med. & Clin. Therapy *8*:375 (Nov.) 1956. 2. Adapted from Hunter, T. H.: J.A.M.A. *144*:524 (Oct. 14) 1950.

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[1] Southworth, J. L. and Dabbs, C. H., "Xylocaine: A Superior Agent for Conduction Anesthesia", *Current Researches in Anesthesia and Analgesia*, May-June, 1953, p. 168.

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